DISEASES TRANSMITTED FROM ANIMALS TO MAN

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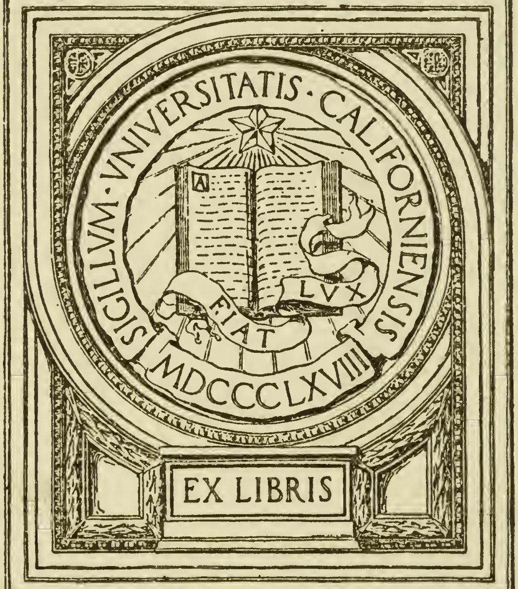

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DISEASES TRANSMITTED
FROM
ANIMALS TO MAN

Diseases Transmitted from Animals to Man

By

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With an Introduction *by*

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TO MY FATHER

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PREFACE

Diseases which may be transmitted from animals to man immediately concern several groups of workers: the veterinarian, the physician, the laboratorian and the health official. Each is engaged with a different phase of the problem however and views the subject from a different angle. My association with these various groups in the past several years has been intimate and cordial, and it has been a pleasure to work with them. The present volume was written with each of them in mind, to afford a common meeting ground where each might understand the problem of the other and thus through concerted effort reduce the number of infections which man contracts from animals.

An attempt has been made to present each disease against a brief historical background, emphasizing its epidemiology and means by which infection may be prevented. Sufficient bacteriology is included to balance the other material. There has been no special effort to include pathology, clinical symptoms or treatment either in man or animals, though of course such items have not been entirely neglected.

The chapter on animal parasites has been written from the standpoint of the public health worker rather than the zoologist. My special thanks go to Dr. W. W. Cort, Dr. Frances A. Coventry and Dr. H. J. VanCleave, who have been of special assistance and have offered constructive criticism.

Acknowledgements are likewise due to numerous friends and associates who have reviewed the manuscript in whole or in part and have given helpful suggestions, especially Dr. C.-E. A. Winslow, Dr. V. A. Moore, Dr. G. W. McCoy, Dr. Edward Francis, Dr. R. R. Spencer, Dr. John R. Mohler, Dr. Adolph Eichhorn, Dr. Henry Field Smyth, Dr. M. P. Ravenel, Dr. I. F. Huddleson, Dr. E. S. Robinson, Dr. F. W. Tanner and Dr. G. E. Gage.

THOMAS G. HULL

September, 1929.

INTRODUCTION

I have a keen appreciation of a book that will bring together available information on the transmission of animal diseases to man. For that reason, I willingly agreed to write an introductory preface to this carefully compiled work of Dr. Hull. There is, in the interest of public health, need for a wider dissemination of existing knowledge of the communicability of certain diseases of animals to man. Further, health officers should recognize more fully than they do the unity of etiology and the multiplicity of ways by which health, either in the individual or in the community, may be impaired. Knowledge of inestimable value for the formulation of health codes is recorded in reports of researches on the nature of animal diseases. This storehouse of information has not been explored as thoroly as it might have been by those who were seeking the cause of many physical ailments, or formulating methods for better sanitation. Milk regulations have emphasized the fundamental principles of cleanliness and low temperatures without considering the physical condition of the cows that produce it. That healthy udders are the first requirement in a safe milk supply is confirmed by recent epidemics of septic sore throat following the use of milk from cows suffering with mastitis.

Comparative etiology teaches that each animal species suffers from infections that have certain analogies with those of man. In some instances several animal hosts may be susceptible to the same infection while in others but a single species is attacked. The latter is the rule but some invading microorganisms and viruses may have several hosts, including man. The anthrax organism and the virus of rabies invade practically all species of warm-blooded animals. The discovery of the transmission of the germ of malta fever from goats to humans and of *Brucella abortus,* the cause of undulant fever, from cattle and swine to man, has explained the sources of two somewhat serious human diseases. Knowledge of their cause and manner of infection suggests ways for their prevention. The studies on an infection in cattle (beef) communicable to man by Gaertner; the researches on swine erysipelas and its transmission to humans by Gerlach;

and the inquiries pointing to the wide distribution of tularemia in the United States by Francis and others, have shown that there is a closer relation between the diseases of animals and those of man than people generally realize. The studies of Robins on glanders in man portrays phenomena of human suffering traced to infection from equines. Nowhere in medical history is there recorded a more horrible situation than that mentioned by Hobday of glanders in children caused by eating the flesh of infected horses.

The many parasites of animals that infest the human species, especially children, render knowledge of the life history of all parasites of animals that invade man a matter of much sanitary importance. The many epidemics of trichinosis, caused by eating infected raw pork, and the prevalence in certain sections of the country of tapeworms, caused by injecting *Cysticercus bovis* in infected beef, point not only to the necessity of extending meat inspection to small cities and towns but also to the importance of properly cooking meat used for food. There is need for a volume that will furnish definite information on the numerous parasitisms that are threatening man because of his association with domesticated animals. Knowledge of the life history and living requirements of such enables one to avoid them.

The assembling of existing facts, relative to the transmission of animal diseases to the human species, will tend to emphasize the importance of competent veterinary service to every community. As the author has pointed out, the rôle played by animals, domesticated by man, in the spread of disease, has shown with great clearness that cooperation between physicians, veterinarians, health officers and live stock sanitarians, especially in the field of preventive medicine, will tend to minimize the dangers from animals on which the American people depend so largely for much of their food and clothing. Physicians and health officers will find much of practical value in this book. It illuminates the value of an understanding of the *symptom-complex* of disease. Someone has said, "There is but one medicine. If there are differences they exist in the species attacked."

VERANUS A. MOORE

CONTENTS

CONTENTS

Preface vii
Introduction ix
Illustrations xv
Tables xvii

SECTION ONE

Diseases of Domestic Animals and Birds

I. Tuberculosis 1
II. Anthrax 39
III. Foot-and-Mouth Disease 53
IV. Malta Fever and Contagious Abortion 62
V. Milk Sickness 86
VI. Actinomycosis 94
VII. Smallpox and Cowpox 100
VIII. Glanders 110
IX. Rabies 118
X. Psittacosis 152
XI. Food Poisoning 155
XII. Swine Erysipelas 170
XIII. Affections Caused by Animal Parasites 175

SECTION TWO

Rodent Affections

XIV. Plague 191
XV. Tularemia 209
XVI. Spirochetal Jaundice 222
XVII. Rat-Bite Fever 231
XVIII. Rocky Mountain Spotted Fever 236

SECTION THREE

Human Diseases Spread by Animals

XIX. The Relation of Human Infections to Animals 247
XX. Septic Sore Throat 250
XXI. Diphtheria 260
XXII. Scarlet Fever 266
XXIII. Other Human Diseases Sometimes Contracted by Animals 271

SECTION FOUR

Animals as Passive Carriers of Disease Organisms

XXIV. Botulism 275
XXV. Tetanus 279
XXVI. Gas Gangrene 285

SECTION FIVE

A Review of the Rôle Played by Each Animal in the Spread of Disease

XXVII. The Rôle of Cattle, Horses, Swine, Sheep, Goats, Dogs, Cats, Rats and Mice, Poultry, Birds, and Wild Game 293
Author Index 307
Subject Index 327

ILLUSTRATIONS

FIGURE

1. *Map showing extent of bovine tuberculosis in the United States* 9
2. *Map showing extent of avian tuberculosis in the United States* 21
3. *Mobile laboratory for inspection of milk-pasteurizing plants* 30
4. *Stamp used by meat inspectors of the United States Department of Agriculture* 32
5. *Federal inspectors making final examinations of beef carcass* 33
6. *Lesions on hoofs in foot-and-mouth disease* 56
7. *Lesions on tongue in foot-and-mouth disease* 58
8. *Map showing world distribution of undulant fever in man, of goat origin* 68
9. *Map indicating origin of discovery of organisms of undulant fever and contagious abortion* 71
10. *Map indicating extent of contagious abortion in cattle in the United States* 74
11. *Lesions in guinea pigs caused by Brucella abortus* 77
12. *Map showing prevalence of contagious abortion among hogs in the United States* 81
13. *Rayless goldenrod* 88
14. *Ophthalmic mallein test for glanders in a horse* 115
15. *Distribution of rabies in the United States in 1926* 122
16. *Distribution of rabies in the United States in 1921* 123
17. *Proper type of dog muzzle* 144
18. *Improper type of dog muzzle* 145
19. *Plague among the Philistines* 192
20. *An ancient conception of the cause of plague* 193
21. *Map showing distribution of plague throughout the World in 1926* 196

22. *Section of tenement house showing conditions that favor the breeding of rats* 200
23. *Ground squirrel* 206
24. *Distribution of tularemia in man in the United States* 210
25. *Jack rabbit* 213
26. *Rabbit's liver in case of tularemia* 214
27. *Ulcer of right finger, with axillary abscess, in case of tularemia* 216
28. *Tularemia due to tick bite* 219
29. *Map showing distribution of Rocky Mountain spotted fever in the United States* 237

TABLES

1. *Susceptibility of certain animals to infection with three types of tubercle bacilli* 6
2. *Human and bovine tubercle bacilli in cases examined in 1910, Park and Krumwiede* 12
3. *Cases infected with bovine tubercle bacilli, compiled from British Royal Commission. Reports by Eastwood and F. and A. S. Griffith, 1914* 14
4. *Human and tubercle bacilli in man, combined table of all cases, Wang, 1917* 14
5. Cases of tuberculosis in England, by age groups and *types of infection. Investigations to 1920, A. S. Griffith* 15
6. *Bovine infection in human tuberculosis in England and Wales. Cobbett, 1922* 15
7. *Results of typing tubercle bacilli from various sources, Van Es* 16
8. *Tubercle bacilli in the feces of cows, Williams and Hoy* 17
9. *Incidence of tubercle bacilli in milk 1893 to 1925,* Tonney, White, and Danforth 19
10. *Swine retained for tuberculosis, Chicago market 1908-1927* 26
11. *Progress of work of establishing accredited herds free of tuberculosis, 1918-1925* 29
12. *Bovine and human types of tubercle bacilli in lymph nodes and meningitis before and after pasteurization of milk supply, New York City* 31
13. *Thermal death points of tubercle bacilli, Park* 32
14. *Diseases and conditions for which condemnations of meat animals were made on post-mortem inspection, 1926* 34
15. *Human cases and deaths of anthrax occurring in man in the United States* 44

16. *Location of lesions in 114 cases of cutaneous anthrax in Massachusetts* 45
17. *Cases of anthrax by occupation, Andrews* 45
18. *Deaths from anthrax in United States registration area by occupation, 1910-1917* 46
19. *Cases of anthrax by occupation, Smyth, 1919-1927* 47
20. *Modes of infection of foot-and-mouth disease, California, 1925* 61
21. *Susceptibility of animals to Brucella melitensis and Brucella abortus* 69
22. *Actinomycosis in the United States, Sanford and Voelker* 98
23. *Pasteur's original schedule of antirabic vaccination* 130
24. *Schedule of anti-rabic vaccination at Pasteur Institute, Paris, in 1927* 131
25. *Persons vaccinated against rabies, with mortality, at Pasteur Institute, Paris* 132
26. *Animals sickened and died, or killed on account of rabies in Prussia* 133
Biting animals, rabid or supposedly rabid, in cases from Paris and Budapest Institutes 134
27. *Comparative report on 825 animal heads received at Illinois State Public Health Laboratory, 1924-1927* 134
28. *Comparative report on 1,494 animal heads received in Oklahoma State Public Health Laboratory, 1925-1926* 135
29. *Human death from rabies in the United States* 139
30. *The rôle of the stray dog in spreading rabies* 148
31. *Isolation of Salmonella organisms in various epidemics of food poisoning, Savage* 160
32. *Vehicles of food poisoning in 100 British outbreaks, 1923-1925* 162
33. *Vehicles of food poisoning in 1,174 alleged outbreaks in the United States, 1910-1925* 163
34. *Organisms of the Salmonella group and their relation to disease in man and animals* 166
35. *Human cases of bubonic plague in the United States* 195
36. *States in which tularemia has been found, with date of first known cases* 212

37. *Rats from different localities infected with Leptospira icterohemorrhagiae* 227
38. *Prevalence of hemolytic streptococci in high grade milk, Frost and Bachman* 253
39. *Septic sore throat in Massachusetts, Bigelow and Forsbeck* 255
40. *Epidemic of septic sore throat at Lee, Massachusetts* 256
41. *Epidemics of milk-borne septic sore throat, 1918-1927* 257
42. *Epidemics of milk-borne scarlet fever* 268
43. *Relative frequency of infection with the Welch bacillus group in war wounds* 286

Section One

DISEASES OF DOMESTIC ANIMALS AND BIRDS

Section One

DISEASES OF DOMESTIC ANIMALS AND BIRDS

CHAPTER I

TUBERCULOSIS

Tuberculosis is a communicable disease caused by some strain of the tubercle bacillus. Man, animals and birds are susceptible to the specific organisms pathogenic of each particular group, but cross infections may occur upon occasion by the organism of another group.

Tuberculosis has been known by a variety of names at different times and under different conditions. Consumption, phthisis and scrofula have been applied to the disease in man and animals alike. "Pearly disease" refers to infection of the serous membranes of cattle.

History

The origin of tuberculosis probably goes back to those dim ages when men began to congregate themselves in compact social groups. The earliest records available indicate that tuberculous infection was wide spread in both man and domestic animals.

The study of Egyptian mummies has shown that the disease was very prevalent in the time of the Rameses thirteen centuries before Christ. Likewise in India the Hindus, in their early writings contained in the Veda in the 15th Century, B.C., are said to have made reference to the disease, as well as the Parsees about the same time. From that time down through the years wherever the white man has left his record, accounts of the ravages of tuberculosis may be found under one name or another. Hippocrates in the fifth century, B.C., encountered it in Greece; Aretaeus in the first century, B.C., recognized its presence in Cappadocia in Asia Minor; the Goths are said to have declared it a contagious disease.

Tuberculosis in animals was likewise recognized in very early times. The Mosaic Law prohibited the use of the flesh from animals suffering from "wen or scurvy." The Talmud of the first century, A.D., the Mishna of the third century and the Gemara of the fifth century, also contain similar prohibitions. Hoare states that in the ninth century the Franks had ecclesiastical laws forbidding the use of the flesh of cattle and pigs with tuberculosis of the serous membranes. In 1370 and 1582 laws dealing with the same subject were enforced also in Germany. In 1702 tuberculosis in cattle was confused with syphilis in man under the name "French disease," resulting in compulsory slaughter of all infected cattle. In 1783 the two types of infection were recognized, resulting in a complete relaxation by the Berlin health authorities of restrictions on meat inspection. About this time, however, the contagiousness of tuberculosis began to be recognized, especially by Rühling in Germany (1774) and Huzard in France (1790). The latter believed that the disease of man and animals was identical, as did also Gürlt (1831) and Spinola (1858). This view was not generally held by most workers at this time; it was regarded as a "fatal thought." Pidoux declared that tuberculosis was "the common results of a quantity of divers external and internal causes, not the product of a specific agent, ever the same" (Valery-Radot).

Observations in human tuberculosis meanwhile had been in progress, which were to lay a sure foundation upon which to build. In 1782 Reid and a little later Baillie (1793) in England called attention to the fact that tubercles increase in size and become purulent, forming lung abscesses. In 1810 Bayle in France differentiated miliary tuberculosis from the pulmonary type. Laennec, a victim of the disease himself, lived long enough to prove the disease was probably communicable and was caused by a specific micro-organism, whatever part of the body was attacked.

Villemin in 1865 presented a paper relating the transfer of infection to rabbits and guinea pigs of both human and animal tuberculosis. He was violently attacked for upsetting all former ideas of infection, but his work was soon confirmed by numerous investigators in France and Germany and England. The germ theory of disease was then demonstrated by Pasteur and research developed rapidly.

Robert Koch in 1882 reported his findings which established beyond any doubt that the cause of tuberculosis was an organism which could be demonstrated under the microscope from tuberculous animals, cultivated in pure culture, made to reproduce the disease in healthy animals and again recovered. Thus were all postulates fulfilled.

In 1889 Rivota and 1890 Maffucci showed that tuberculosis of birds was caused by an organism slightly different but closely related to the mammalian organism. In 1896 Theobald Smith proved that human and bovine tuberculosis were not caused by identical organisms but by separate although closely related varieties.

Types of Organisms

Three types of tubercle bacilli concern man—the human, the bovine and avian. All three are without doubt members of a common parent strain, but which through long years of adaptation to different hosts have acquired particlar biological characteristics.

Mycobacterium tuberculosis (hominus)
The tubercle bacillus, human type.

Mycobacterium tuberculosis (bovis)
The tubercle bacillus, bovine type.

Mycobacterium avium
The tubercle bacillus, avian type.

The human type of tubercle bacillus finds it chief reservoir of infection in man, but upon occasion it infects certain animals and birds. It will be discussed here, however, only as animals and birds act as agents for the transfer of the disease back to man. It has no outstanding morphological characteristics which can be relied upon to distinguish it from the bovine or avian strains. Culturally it differs from the others in several respects, chief of which is its growth on glycerin media as contrasted to the bovine organisms and its preference for human bile rather than ox bile. From the pathogenic standpoint the human type can be readily distinguished by its virulence for guinea pigs and its almost total lack of pathogenicity for rabbits and fowls.

The bovine type of tubercle bacillus infects practically all animals as well as certain birds and man. It has no constant distinguishing morphological characteristics. In early cultures it will not grow well upon glycerin media while the human type grows very well; it prefers ox bile to human bile media. Its virulence for rabbits is marked and constant while it is without pathogenic powers for fowls. Calmette states however that as a matter of practical application, diagnosis for the bovine bacillus can be established only by inoculation of a calf or by mammary infection of a lactating goat.(1)

The avian type of tubercle bacillus has acquired rather distinctive characteristics through long adaptation in a host of higher body temperature than man or animals. The morphological characteristics of the organism are no different from the human type. Culturally it is different from both the human and bovine in that it will grow in original culture upon plain agar. While its optimum temperature for growth is 40°C. to 43°C. it will grow at a temperature as low as 28°C.; neither of the other strains will grow below 36°C. Its special pathogenic characteristic is its fatality for all fowls and domestic birds.

Cold-blooded animals are subject to a disease simulating tuberculosis but caused by saphrophytic organisms which bear little relation to the tubercle bacilli of warm-blooded animals. Fish, frogs, lizards, turtles, toads and snakes have been found susceptible to infection.

Bergey lists the following organisms as members of this group (2):

Mycobacterium piscium
The cause of nodule and tumor-like formations in carp, infectious also for frogs and lizards but not for guinea pigs or pigeons.

Mycobacterium ranae
Isolated from the liver of frogs, infectious for all cold blooded animals.

Mycobacterium tropidonatum
The cause of nodules and tumor-like formations in snakes.

Mycobacterium chelonei
Commonly known as the turtle bacillus. Isolated from lungs of turtles and infectious for all cold-blooded animals, but not for warm-blooded with the exception of the guinea pig.

The organisms are acid-fast and morphologically resemble tubercle bacilli. They grow abundantly on all culture media, however, and are without pathogenic properties for warm-blooded animals. They are incapable of serving as antigen in the complement fixation test with tuberculous serum, thus differentiating them serologically. Extracts from the organisms, made after the manner of tuberculin, are without toxic effects upon tuberculous animals in large amounts, whereas small amounts of tuberculin are fatal. The bacillus of Friedmann, isolated from a turtle, was proposed as a vaccine for the prevention and cure of tuberculosis in cattle and man. No protection could be demonstrated after extensive experiments however. Numerous attempts have been made by various investigators to identify the tubercle bacilli of animals and birds with the acid-fast organisms of cold-blooded animals but to no avail. They are distinctly different organisms.

Several other acid-fast bacilli belong in the group of mycobacteria, which have little significance in the relationship of animal diseases to man. Johne's bacillus *(Mycobacterium paratuberculosis)* is the cause of chronic dysentery of cattle. Although originally thought to be a form of intestinal tuberculosis, later workers showed that Johne's disease was a separate infection. The organism has never been found to infect man. The mist bacillus, *Mycobacterium stercusis,* found in cow dung, is fatal to guinea pigs and causes a disease in rabbits simulating tuberculosis, but is without pathogenic properties for man. The butter bacillus *(Mycobacterium butyricum)* causes a benign infection in guinea pigs; the smegma bacillus *(Mycobacterium smegmatis),* a saprophyte of man, likewise causes a slight infection of guinea pigs; the timothy bacillus *(Mycobacterium phlei),* a saprophyte of the hay loft, is not unlike the mist bacillus in its pathogenic properties.

Others of the group include *Mycobacterium berolinensis* and *Mycobacterium friburgensis,* both isolated from butter, *Mycobacterium graminis* of plant dust and *Mycobacterium alluvialum* from the soil.

Since all of these organisms are harmless for man, they could be entirely ignored were it not for the fact that morphologically they resemble true tubercle bacilli and hence at times cause confusion in diagnosis.

SUSCEPTIBILITY TO INFECTION

Tuberculosis is one of the penalties of civilization, whether in man or animals. The disease is unknown among the wild tribes of the earth until they come into contact with the outside world; the infection then spreads like wild-fire, causing great havoc. Likewise animals in the wild state never contract tuberculosis spontaneously; when confined in menageries and zoos they often succumb. Wild species of birds are free of infection except such carnivores which come into contact with domesticated diseased animals or birds; in aviaries, tuberculous infection is prevalent as it is in domestic poultry.

Man is much more susceptible to the human type of tubercle bacillus than to either the bovine or avian. The bovine bacillus has a predilection for the glands and the intestine although the pulmonary form has been encountered, as well as infections of the bone and joints, tuberculous meningitis and generalized tuberculosis. Human adults are practically immune to infection with the bovine bacillus but small children are quite susceptible. Park found 111 bovine infections among 1220 cases of tuberculosis, 101 of which were in children under 16 years of age.

The avian tubercle bacillus for many years was thought to be non-virulent for man but a score or more of cases have recently been compiled, due to this type of infection. (See Avian Tuberculosis.)

There is a very general agreement throughout the world that practically all infection whether of human or bovine origin, takes place during infancy and childhood.

The anthropoid apes and monkeys are extremely susceptible to tuberculous infection when confined in menageries, spontaneous infection taking place with either the bovine or human types of organism. Rabinowitsch (3) found the human type in 19 out of

Table 1

SUSCEPTIBILITY OF CERTAIN ANIMALS TO INFECTION WITH THREE TYPES OF TUBERCLE BACILLI

(After Cobbett, Revised) Severity of Disease by Type of Tubercle Bacilli

	Human	*Bovine*	*Avian*
Man	++	+	±
Anthropoid apes and monkeys	+++	+++	?
Dog	+	+	0
Guinea pig	+++	+++	+
Rabbit	±	+++	++
Hog	++	++	++
Horse	0	+	±
Cow	±	++	±
Goat	±	++	±
Sheep	±	±	±
Cat	±	++	±
Rat	+	+	+
Mouse	+	+	+
Wild animals in captivity	±	++	?
Parrot and cockatoo	++	++	++
Fowls and other domestic birds	0	0	++

27 monkeys dying in a zoological garden, while only 3 showed the bovine type. Nocard, (4) however, demonstrated that when equal amounts of both types of organisms were administered by way of the mouth, the bovine bacillus was the more virulent.

The domestic cow is infected with tuberculosis of the bovine type in amounts varying from 50 per cent in the specialized dairying districts down to less than half of one per cent in the isolated range districts. The human type of bacillus has never been found to infect cattle spontaneously. When injected under the skin it forms but a local lesion which eventually heals. The avian bacillus may infect calves which live in quarters to which infected poultry have access. The virulence of the organism however, is slight. The goat has been generally supposed by popular opinion to be immune to tuberculosis. This notion has been used to promote the sale of goat's milk as free from danger of harboring tubercle bacilli. A considerable number of instances are on record, however, of infection from such a source, the bovine bacillus being the causative organism. The human as well as the avian bacillus has no more than a local effect upon the animal. Tuberculous infection of goats is not wide spread, due to the liberty such animals usually have; when confined in stables with tuberculous cows, however, infection may take place.

Sheep are rarely found infected, but are susceptible to the bovine form of the disease especially when confined in stables. The human and avian types are of small significance. Hogs are susceptible to infection with all three types of tubercle bacilli. While it was originally thought that the bovine form of the organism was responsible for practically all tuberculosis in swine, it was found that hogs from "accredited areas," where the disease in cattle had been eliminated, still continued to show infection in considerable numbers. A careful study showed that infection was due to the avian bacillus. Hogs fed on garbage, as from a tuberculosis sanitarium, may contract the human type of disease.

The horse is infected but rarely with tuberculosis. Calmette states that the disease was found but once in 15,000 animals at the abottoirs in France. Other investigators have found horses infected at infrequent intervals, usually with the bovine bacillus, but occasionally with the human organism. The avian bacillus has been found a few times. Since the lesions are usually abdominal, ingestion seems to be the method of infection, either from drinking cow's milk as colts or from living in close contact with infected cattle. Hoare (6) believes that tuberculosis in the horse is much more common than ordinarily believed, the lesions usually being overlooked.

Donkeys and mules are apparently quite immune to the disease.

The dog contracts bovine tuberculosis from ingesting infected meat or milk. The human type of the disease is said to be more common through close association with tuberculous persons. In Europe Douville (7) found about 4 per cent of 20,000 dogs infected. From a careful study of the type of infecting organism it would appear that three cases due to the human bacillus are found to each one of bovine. Dogs are immune to avian infection.

Cats are infected somewhat more commonly than dogs, especially with bovine bacilli from infected milk. Rabinowitsch, in examining six cats found three human type organisms and two bovine type.

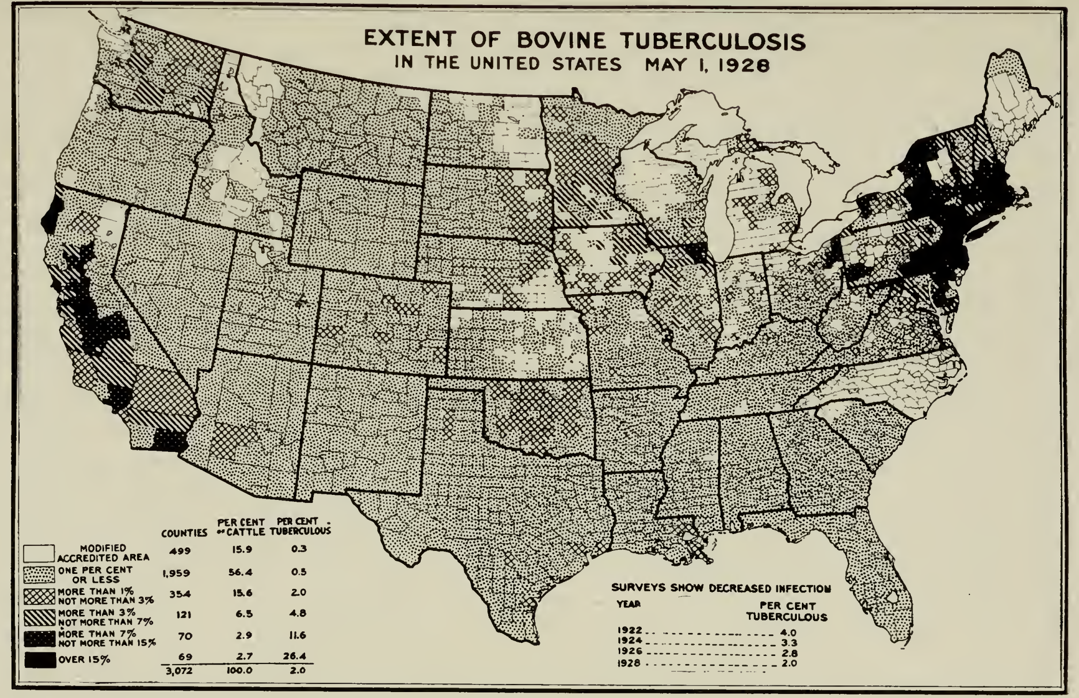
Rabbits are immune to human tubercle bacilli except for a local lesion, but succumb easily to injection of the bovine or avian organism. They are rarely known to contract the disease spontaneously, however, either in the wild state or in hutches. Guinea pigs may be infected easily by injections of either the human or bovine bacilli, but are rather more resistant to the avian strain. Rats and mice may contract the three forms of the disease either spontaneously or artificially.

Birds of all kinds are susceptible to the avian type of tubercle bacillus. With the exception of the cockatoo and parrot, however, they are entirely resistant to human and bovine strains.

Bovine Tuberculosis

It is not certain when bovine tuberculosis was introduced into the United States, but it is not improbable that it was in the early days of the colonies through the importation of infected cattle. It is stated, however, that the disease was rarely seen in the United States before 1870. (8) Importation of better grades of cattle from Europe where the infection was very high, served to increase the spread.

Because of the chronic course of the disease, the notion arose that is was not communicable; breeders, therefore, sold their cattle as soon as they discovered the existence of the malady. In this way bovine tuberculosis was spread before its infectious nature was realized.


EXTENT OF BOVINE TUBERCULOSIS
IN THE UNITED STATES MAY 1, 1928
COUNTIES
PER CENT OF CATTLE
PER CENT TUBERCULOUS
MODIFIED ACCREDITED AREA 499 15.9 0.3
ONE PER CENT OR LESS 1,959 56.4 0.5
MORE THAN 1% NOT MORE THAN 3% 354 15.6 2.0
MORE THAN 3% NOT MORE THAN 7% 121 6.5 4.8
MORE THAN 7% NOT MORE THAN 15% 70 2.9 11.6
OVER 15% 69 2.7 26.4
3,072 100.0 2.0
SURVEYS SHOW DECREASED INFECTION
YEAR
PER CENT TUBERCULOUS
1922 4.0
1924 3.3
1926 2.8
1928 2.0

United States Department of Agriculture

Fig. 1.—Map showing extent of bovine tuberculosis in the United States.

The first exact figures as to the prevalence of the disease came with the introduction of the use of tuberculin. From 1893 to 1908 about 400,000 tuberculin tests of cattle were made in the United States, of which 37,008 or 9.25 per cent were positive. In 1909 a program showing the value of tuberculin as a means for tuberculosis eradication was begun in Washington, D.C. Of 1,701 cattle in the District of Columbia, 18.87 per cent reacted; in 1920-21 there were 1,313 cattle in the same area, only 5 of which reacted, (0.4 per cent).

Dairy cattle are much more heavily infected than beef cattle, varying from 5 per cent in the sparsely settled regions to 50 per cent in the centers of heavy milk production, with an average of 10 per cent for the country. Beef cattle on the other hand are usually slaughtered young, less often stabled and contain a greater proportion of males so that the lowering of vitality by milk secretion plays a smaller role. Tuberculous infection varies from 0.14 per cent to 30 per cent, with an average of about 2 per cent for the country.

Tuberculosis in its early stages in the cow, as in man, usually passes entirely unnoticed. The cough, fever, loss of flesh and other symptoms are not present for a considerable period of time, until the disease has advanced to its later stages.

Infection of the cow may take place by inhalation into the lungs, by ingestion with infected or contaminated food, by sexual contact with an animal having infected sexual organs, or from the tuberculous mother through the fetus.

The organisms upon entrance localize in various parts of the body where they set up pathological processes more or less extensive. In the majority of cases the disease is limited to the lungs and the serous membranes of the thorax and abdomen. Other organs, however, such as the liver and spleen may be infected. Involvement of the various organs has been given by Ostertag on 43,000 observations made at the German slaughter houses.

	Per cent infected		*Per cent infected*
Lungs	75.0	Heart	0.9
Visceral pleura	55.0	Kidneys	0.7
Peritoneum	48.0	Bones	0.4
Costal pleura	7.0	Diaphragm	0.2
Liver	28.0	Larynx	0.13
Spleen	19.0	Brain	0.04
Trachea	3.0	Spinal cord	0.03
Intestine	1.0	Tongue	0.01

Figures compiled in Bavaria and Baden (cited by Mohler) show a somewhat different distribution.

Bavaria:	*Per cent*
Tuberculosis of lungs and serous membranes	41
Tuberculosis of lungs alone	33
Tuberculosis of serous membranes alone (pearly disease)	17
Tuberculosis of other organs	8

Baden:	*Per cent*
Tuberculosis of lungs alone	21
Tuberculosis of serous membranes alone	28
Both combined	39
Generalized tuberculosis	9
Tuberculosis of the sexual organs alone	3

Tuberculous infections of the serous membranes is often known as "pearly disease" because of the peculiar appearance of the tubercles. Lesions of the udder have a particular significance for the public health because of the enormous numbers of tubercle bacilli that may enter the milk directly. Usually only one quarter of the udder is involved, rarely more than two, and almost always the hind quarters. In the early stages, at least during the first month, the lesions are not of sufficient extent to attract notice, although tubercle bacilli may be excreted. Later, the udder becomes hard and large, secreting a watery milk mixed with flakes and tubercle bacilli.

Human Infections with Bovine Tubercle Bacilli

The positive proof that bovine tubercle bacilli were pathogenic for man was first demonstrated by Ravenel from the State Livestock Sanitary Board of Pennsylvania in 1901. In a paper before the London Congress on Tuberculosis in 1901 he cited five such human infections, concluding:

> "It is a fair assumption from evidence at hand, and in the absence of evidence to the contrary, that the bovine tubercle bacillus has a high degree of pathogenic power for man also, which is especially manifest in the early years of life." (34)

A formidable list of human cases infected with the bovine tubercle bacillus has been assembled by various investigators. In 1902 Kober (9) compiled the records of 86, this being all such published in scientific literature up to that time. Bacteriological evidence as to the type of infection was lacking of course, since at that time laboratory methods had not been perfected sufficiently to distinguish between human and bovine organisms.

Table 2

HUMAN AND BOVINE BACILLI IN CASES EXAMINED IN 1910
(Park and Krumwriede)

Diagnosis of cases examined	Adults 16 yrs. & over		Children 5 to 16 yrs.		Children Under 5 yrs.		Notes
	Human	*Bovine*	*Human*	*Bovine*	*Human*	*Bovine*	
Pulmonary tuberculosis.	278		8		5		Clinical diagnosis only known, and therefore no positive details as to extent of tuberculosis elsewhere than in lungs.
Tuberculous adenitis, inguinal and axillary...	1		4				See next
Tuberculous adenitis, cervical............	9		19	8	6	12	In two cases cultures were from axillary node, but primary focus was cervical. Another case died shortly afterward with pulmonary tuberculosis.
Abdominal tuberculosis..	1		1	1		3	Milk-supply of one child subsequently examined. Tubercle bacilli isolated.
Generalized tuberculosis, alimentary origin....	2		1		12	4	One bovine case had tuberculous osteomyelitis of metatarsal bone.
Generalized tuberculosis including meninges...					18	1	
Tuberculous meningitis..			1		14	1	No autopsy. Extent of lesions elsewhere unknown.
Tuberculosis of bones and joints..........	1		10		6		
Genito-urinary tuberculosis...............	3	1	1				The adult bovine case was tuberculosis of one kidney. Removal of kidney. Complete recovery.
Tuberculous abscesses...	1						Possibly primary in bone.
Total..................	296	1	45	9	62	22	

In 1910 Park and Krumwiede (10) tabulated 436 cases studied by themselves, regarding the relative importance of the bovine and human types of tubercle bacilli in different forms of human tuberculosis. Thirty-two cases showed the bovine type of organism (Table 2).

The next year they combined their own tabulations with those of various other workers both in America and Europe, obtaining a total of 1,224 cases, of which 111 were bovine origin.

A. S. Griffith (12) in 1914 published figures showing that out of 65 deaths from all causes in England, 6 were due to the bovine type of tubercle bacillus, 20 to human and 1 mixed. In another group from a tuberculosis institution, 6 deaths were due to the bovine type of bacillus, 28 human and 1 mixed. A compilation of the reports and papers published by Eastwood and F. and A. S. Griffith showed that 17.6 per cent of the deaths of 113 children under 12 years were due to the bovine type (table 3).

In 1917 Wang (11) published a summary of all reports which had appeared up to that time on the relation of the bovine tubercle bacillus to man. Besides 88 of his cases (1917), there were 72 cases of Mitchell (1914), 70 cases of Fraser (1912), 430 cases of Stanley Griffith (1914, 1915-17, 1916), 430 cases of Eastwood and Fred Griffith (1914, 1916) and 1511 cases collected by Park and Krumwiede (1912) including those of the British Royal Commission (1907-11) and Bullock (1910).

Of the tuberculous children under 5 years of age, 32.4 per cent were infected with the bovine organism, against 2.9 per cent bovine infection in tuberculous adults (table 4).

In 1922 Cobbett (5) collected all cases of tuberculous infection occurring at all ages in England and Wales, finding 7 per cent were due to the bovine bacillus (table 6).

In 1923 Gordon and Brown (14) found the bovine organism 10 times among 30 children under 12 years of age. Among 23 under 5 years of age, 6 showed the bovine bacillus, while among the 7 between 5 and 12 years 4 showed the bovine organism. There was only one instance of an infant under 1 year showing such infection.

From a comprehensive review of the subject Kiernan concludes that the bovine tubercle bacillus is less virulent for man than the human tubercle bacillus and that it is erroneous to believe that small doses of living bovine tubercle bacilli taken with food raise the specific resistance of individuals without producing disease.

Table 3

Compiled from British Royal Commission Reports and Papers Published by Eastwood and F. and A. S. Griffith, 1914

Varieties	Number of cases	Percentage of cases infected with bovine bacilli	
		Under 5 years	all ages
Cervical lymph nodes	125	85 .0% of 20 cases	48 .0%
Lupus	140	66 .0% of 50 cases	51 .0%
Scrofuloderma	52	58 .3% of 12 cases	38 .4%
Bone and joint	514	30 .2% of 96 cases	19 .2%
Genito-urinary	21		19 .0%
Meningeal	12		16 .6%
Pulmonary	275		1 .1%
Postmortem cases, children under 12	113	21 .3% of 61 cases	All ages under 12 17 .6%

These cases in children under 12 classified according to anatomical distribution of the primary lesion, showing percentage of bovine infections.

Alimentary	35		80 .0%
Respiratory, double portal respiratory and alimentary and uncertain	116		1 .8%

Table 4

COMBINED TABLE OF ALL CASES

(Wang, 1917)

Classification	Adults 16 years or over		Children 5 to 16 years		Children under 5 years	
	Human	Bovine	Human	Bovine	Human	Bovine
Pulmonary tuberculosis, including sputum	1000	5	28		45	1
Abdominal tuberculosis	24	7	13	17	29	34
Generalized tuberculosis	39	2	32	3	169	22
Genito-urinary	35	4	4			
Tuberculous meningitis	6		13	5	55	10
Skin	12	3	4	6	2	
Cervical adenitis	62	10	61	76	18	75
Axillary adenitis	6		8		4	
Bone and joint	82	4	255	61	89	54
Latent tuberculosis	2	1	2	2	4	1
Miscellaneous (other forms)	5	2		1		2
	1273	38	420	171	415	199
Percentage of bovine infection at each age period	2 .9		28 .9		32 .4	

Total in 2516 of 16 percent bovine type.

Table 5

TABLE SHOWING TOTAL NUMBER OF HUMAN CASES OF TUBERCULOSIS OCCURRING IN ENGLAND

Investigations to 1920, by Age groups.
A. S. Griffith

Age Periods	No. of cases	Types of Infection						Percent of Bovine
		Standard types			Atypical			
		Human	Bovine	Mixed	In culture	In virulence		
					Human	Human	Bovine	
0–5	221	123	76	2	2	3	5	37 .55
5–10	312	208	81	1	5	7	10	29 .45
10–16	150	119	17	–	6	3	5	14 .66
16 & upwards	384	342	20	2	8	10	2	6 .25
Total	1068	803	194	5	21	23	22	20 .7

Table 6

PROPORTION OF BOVINE INFECTION IN VARIOUS KINDS OF HUMAN TUBERCULOSIS AT ALL AGES IN ENGLAND AND WALES

(Cobbett, 1922)

Method of infection	Varieties of tuberculosis	Types of tubercle bacilli (percent)	
		Human	Bovine
Group 1: Aerial infection....	Pulmonary..............	98 .7	1 .3*
	Bronchial glands.........	94 .5	5 .5
Group II: Food...........	Intestinal, peritoneal, mesenteric gland.............	50	50
	Cervical gland...........	54	46†
Group III. Various degrees of dissemination from either aerial or food infection	Generalized tuberculosis...	84	16
	Meningitis..............	82	18
	Bones and joints..........	80	20‡
	Genito-urinary...........	82	18
Group IV. Skin infection ...	Lupus..................	50	50

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* Figures for the whole world show that the percentage of bovine infection in pulmonary tuberculosis is only 0.4.

† Mitchel gives 90 percent for Scottish cases and Griffith 71.4 percent; United States 53 percent.

‡ Fraser gives 58.6 percent for Scottish cases and Griffith, 29.6 percent; world figures 21.6 percent.

Table 7

RESULTS OF TYPING TUBERCLE BACILLI FROM VARIOUS SOURCES

(Van Es).

Origin of Lesions	Mammalian	Bovine	Human	Avian	Mixed	Negative	Total
Bovine Lymphnodes.	46	—	—	7	4	26	83
Bovine—skin.......	—	—	—	—	—	38	38
Bovine—Hemorrhagic lymphnodes of no lesion reactors..	—	—	—	—	—	20	20
Bovine—uteri of no lesions reactors....	—	—	—	—	—	3	3
Human—lesions other than pulmonary...	—	—	22	—	—	6	28
Bovine—pulmonary.	—	—	1		—	—	1
Avian—fowls and turkeys..........	—	—	—	33	1	1	35
	46	—	23	40	5	94	208

Transmission of Tubercle Bacilli Through Milk

There are two methods by which milk may become contaminated by tubercle bacilli. The first is directly from the udder of the cow. In the initial stage udder infection is extremely difficult to diagnose. The animal may show no other signs of tuberculosis than in positive tuberculin reaction, the changes in the udder being only of miscroscopic size. Yet a few tubercle bacilli will pass out into the milk. In the later stages, one or more quarters will become densely swollen and hardened. The milk will have a normal appearance for a time but eventually becomes serous and yellow, containing small purulent lumps. As many as 100,000 tubercle bacilli per cubic centimeter will now be found in the milk. Some question has arisen as to the possibility of a normal udder passing organisms from the blood stream. While under ordinary conditions the normal udder will act as a perfect filter, it is possible that there may be temporary lapses when a few tubercle bacilli may pass into the milk.

The second method is by contamination with cow dung. A cow infected with pulmonary tuberculosis does not expectorate the material coughed from the lungs but rather swallows it. Living tubercle bacilli therefore pass out with the dung. Careless milkers allow more or less of this material to fall into the milk from the udder and sides of the cow thus causing contamination of the milk. The amount of fecal matter which milk will dissolve without apparent sign of such pollution is quite large. Since much of it is soluble the tubercle bacilli are set free and ordinary filtering removes only the larger particles. Williams and Hoy(31) found that even apparently healthy cows excreted in the feces tubercle bacilli pathogenic for guinea pigs, (table 8).

Table 8

TUBERCLE BACILLI IN THE FECES OF COWS

	Cows tested	Tubercle bacilli in feces	Percent Positive	Tuberculin test
Moore & Boynton....	36	0	0	Positive
Peterson............	14	0	0	Positive
Peters & Emmerson...	41	3	7	Positive
Reichel and Deubler..	19	2	10	Positive
Reichel and Deubler..	8	2	25	—
Royal Commission....	3	1	33	Positive
Schroeder & Cotton...	5	1	20	—
Titze and Thieringer..	7	0	0	—
Titze and Thieringer..	38	0	0	Positive
Williams & Hoy......	391	6	1.53	All but 24 negative

Milk from tuberculous cows therefore is always a potential source of danger. Laboratory examination of a single sample of milk from an animal suspected of being tuberculous is of small value in either diagnosing the disease or as a criterion of safety of the milk. Microscopical examination of sediments are misleading because of the possibility of finding harmless acid-fast saprophytes. Animal inoculation requires a period of several weeks. Positive findings are indisputable evidence of tuberculous infection but negative findings only mean that the particular sample of milk examined was not contaminated. The cow may have been infected but failed to pass the organisms through an udder that at another time would be defective as a filter, or the milker in taking the sample was careful to exclude dirt and dung which ordinarily might find its way into the milk.

The extent of contamination of raw milk with tubercle bacilli has been demonstrated by numerous investigators. Tonney, White and Danforth (15) have collected statistics in 16,700 samples of market milk of which 8.66 per cent showed contamination with tubercle bacilli. The figures varied from 61 per cent contaminated in Berlin in 1894 to 3.5 per cent in Chicago in 1923-1925, (table 9). Ordinarily where no precautions are taken to prevent infection about 10 per cent of the milk samples will be found showing contamination. In Washington, D.C., in 1908, 6.7 per cent was found positive, in New York City in 1909, 16 per cent, in Chicago in 1910 10.5 per cent. At the present time there is no reason to believe that the number of infected samples is decreasing except where special programs are initiated to combat the disease. As cities grow larger, the neighboring dairy districts become more and more intensive in the production of milk, thus producing conditions favorable to the spread of tuberculosis. The amount of infected milk which may be brought into a large city is enormous. Tonney, White and Danforth estimated that for Chicago, with its low percentage of 3.5 per cent in 1923-25, there were 43,750 quarts daily or 15,000,000 quarts per annum infected with tubercle bacilli.

Danger from infected milk supplies may be eliminated in either of two ways, both of which however, are in operation. One is the expensive and difficult task of eradicating the disease in the cow; the other is the fairly simple and economical method of pasteurization.

The tuberculosis eradication program is discussed elsewhere (page 28). It is being promoted primarily by the various State and Federal Departments of Agriculture as an economic problem in the dairy industry. More and more however, milk from tuberculous free cows is being specified by city health departments as a health measure. Some years ago such action by health departments caused much antagonism for the simple reason that machinery was not available for putting on a tuberculosis eradication program. Since 1917, as the disease has been eliminated from various herds, it has been possible to get tuberculosis-free milk in quantities large enough to supply whole cities, and dairymen whose herds have been cleaned up naturally welcome such action by health authorities. In 1926 when the Chicago Health Department specified that all milk coming into that city should be produced by cows free of tuberculosis, there was much criticism for a time by the dairy interests, not because of antagonism to the program, but because of the short period given in which to meet the requirements. It will be many years however, before bovine tuberculosis is entirely eradicated from dairy cattle, and as long

Table 9

CHRONOLOGICAL TABLE SHOWING INCIDENCE OF TUBERCLE BACILLI IN MARKET MILK

Reported from 1893 to 1925 inclusive. (Tonney, White, and Danforth)

Observer	Date	Place	Kind of sample	No. of sample	No. Positive	Percent Positive
St. Friis	1893	Copenhagen	raw	28	4	14.3
Schroeder	1893	Washington	raw	19	1	5.3
Obermueller	1894	Berlin	raw	13	8	61
Ernst	1895	Boston	raw	13	3	23.1
Zacharbekoff	1895	St. Petersburg	raw	22	2	9.1
Buegge	1896	Halle	raw	6	2	33.3
Rabinowitsch	1897	Berlin	raw	25	7	28
Delepine	1896–98	Liverpool	raw	224	28	12.5
Boyce, Hamilton, Woodhead, Delepine	1897–99	Liverpool	raw	545	49	9
Massone	1896	Genoa	raw	33	3	9
Ott	1897	Schwabisch Gmund	raw	28	3	10.7
Petri	1898	Berlin	raw	64	9	14
Jaeger	1899	Konigsberg	raw	6	2	33.3
Kanthack & Sladen	1899	Cambridge	raw	16	9	56.3
Ascher	1899	Konigsberg	raw	17	1	5.9
MacFadyean	1899	London	raw	77	17	22
Pawlowsky	1900	Kiev	raw	51	1	2
E. Klein	1900	London	raw	100	7	7
Max Beck	1900	Berlin	raw	56	17	30
Santori	1900	Rome	raw	90	4	4.4
Hope	1896–03	Liverpool	raw	2885	128	4.4
Klein	1904–06	London	raw	64	5	7.8
Collingridge	1905	London	raw	22	2	9
A. Eber	1905–06	Leipzig	raw	210	22	10.5
Proskauer, Croner Seligman	1905	Berlin	raw	22	10	45.4
E. Prussian Dutch Held Brook Society	1906 and yrs. previous	Konigsberg	raw	1596	97	6.1
Anderson	1908	Washington	raw	232	16	6.9
Mohler	1908	Washington	raw	73	2	2.7
Schroeder	1908	Washington	raw	36	2	5.5
Stokes	1907–09	Baltimore	raw	88	4	4.5
Hess	1909	New York	(raw	107	17	15.9
			(Past	8	1	12.5
Campbell	1909	Philadelphia	(raw	123	18	14.6
			(Past	12	1	8.3
Goler	1909	Rochester N.Y	raw	80	5	6.3
Tonney	1910	Chicago	raw	95	10	10.5
Eber	1908–10	Lautenthal in Harz	raw	158	4	2.53
Thöni	1912–13	Berne	raw	212	17	8
Mitchell	1913–14	Edinburgh	raw	406	82	20
Miller	1913	Edinburgh	raw	101	13	12.9
Delepine	1897–17	Manchester	raw	8040	736	9.15
Bartlett	1923	New Haven	raw	51	18	44
Wilson and Nutt	1925	Manchester	raw	398	52	13
Tonney, White, Danforth	1923–25	Chicago	raw	258	9	3.5
Total...........	....	....	.....	16,700	1448	8.66

as foci of infection exist, the potential danger of reinfection of healthy herds cannot be ignored.

Transmission of Tubercle Bacilli Through Meat

The flesh of tuberculous animals has been considered dangerous for a long time. In 1868 Chauveau showed that by feeding such material to other animals, tuberculosis could be produced.

Tubercle bacilli may sometimes exist in the organs or the muscle tissue in considerable abundance, not only when apparent lesions exist but also when the tissue and organs show no macroscopic evidence of the disease, as was demonstrated by Bongert and other workers in Germany. The question arises therefore whether a tuberculous animal should be entirely rejected for food purposes or whether certain parts may be safely used for human consumption.

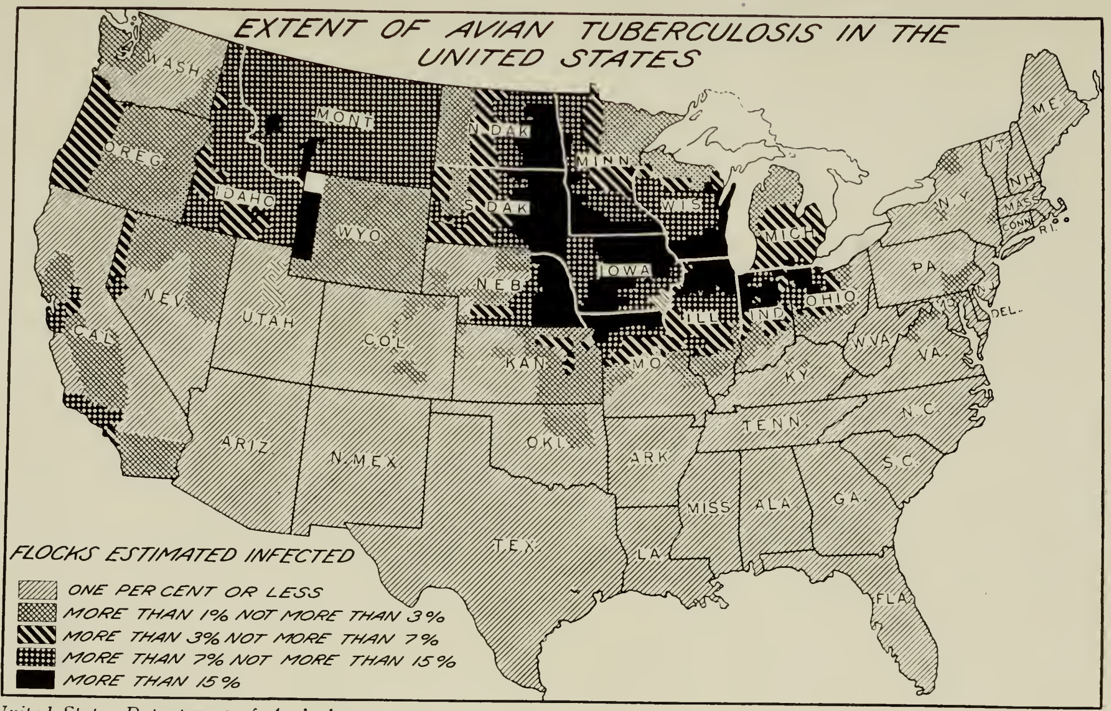
In general it may be stated that muscle tissue is but rarely the seat of tuberculosis (although the closely associated lymphatic glands may be infected). The carcass of a beef animal, if otherwise healthy, may be used for food, therefore, if the lesions are confined to the lungs, liver and thoracic and pharyngeal lymphatics. On the other hand the entire carcass should be discarded should the lesions be present in the pleura, peritoneum, the muscular system including the glands between the muscles, or if there is miliary tuberculosis of the lungs. The pig shows a greater tendency to generalized tuberculosis and greater care must be exercised when these animals show any signs of tuberculosis.

Avian Tuberculosis

The early bacteriolgists isolated tubercle bacilli in pure culture from birds and poultry but it was not not until 1889 that Rivolta showed the avian type to be distinct from both the human and bovine types. This work was immediately confirmed and accepted by other investigators.

The economic losses due to the disease in poultry run into millions of dollars annually and the disease seems to be increasing. The federal department of agriculture, together with the various states, is making a careful study of the disease and attempting its elimination.

For the year ending June 30, 1928 there were 230,000 flocks containing 21,000,000 birds inspected in the United States, of which 16,000 reacted.


EXTENT OF AVIAN TUBERCULOSIS IN THE UNITED STATES
WASH.
OREG.
CAL.
IDAHO
NEV.
MONT.
WYO.
UTAH
ARIZ.
COL.
N. MEX.
N. DAK.
S. DAK.
NEB.
KAN.
OKL.
TEX.
MINN.
IOWA
MO.
ARK.
LA.
WIS.
ILL.
MISS.
MICH.
IND.
OHIO
KY.
TENN.
ALA.
GA.
FLA.
S.C.
N.C.
VA.
W.VA.
PA.
N.Y.
N.J.
MD.
DEL.
VT.
N.H.
ME.
MASS.
CONN.
R.I.
FLOCKS ESTIMATED INFECTED
ONE PER CENT OR LESS
MORE THAN 1% NOT MORE THAN 3%
MORE THAN 3% NOT MORE THAN 7%
MORE THAN 7% NOT MORE THAN 15%
MORE THAN 15%

United States Department of Agriculture

FIG. 2.—Map showing extent of avian tuberculosis in the United States.

The first outbreak of avian tuberculosis in the United States was reported from Oregon in 1900 (16). Since that time the disease has gradually been found in other parts of the country until it is almost universal in extent. The heavily infected areas, however, are the northern middlewest and northwest states. One cause for the increased incidence of the disease in these sections may be that poultry raising is secondary. Hens may be kept till they are four or five years old, allowing plenty of time for tuberculosis to develop. In the eastern states, where the farms are smaller, poultry raising is more commercialized. Only pullets are kept, since they show greater profits during the first year of egg production. It has been found that only 1.5 per cent of pullets give positive tuberculin reactions, whereas 20 per cent of older birds give such a reaction.

Infection in birds may locate in the spleen, liver or lymph nodes of the abdominal cavity. Infection of the lungs is rare. Lesions of the intestinal wall eventually break down, releasing enormous numbers of organisms into the intestine to be eliminated with the feces. The usual manner in which infection is spread from bird to bird is through the feces, either by ingestion of infected material or by scratching the eye or the beak with a contaminated claw. Calmette was successful in causing tuberculosis in birds by placing a single drop of avian tubercle bacilli in the eye.

Eggs from infected hens may contain tubercle bacilli at times. Fitch (17) observed sixty-two tuberculous hens, finding that nineteen (30 per cent) produced no eggs at all. A total of 876 eggs from the other hens were examined for tubercle bacilli, the organisms being found in the composite of nine eggs. It would appear from this work that avian tubercle bacilli are present in less than one per cent of the eggs from tuberculous fowls. An attempt to demonstrate tubercle bacilli on the shells of eggs from infected hens was entirely unsuccessful. Two hundred and nine eggs from tuberculous birds in no instance showed tubercle bacilli on the shells.

Eggs containing avian tubercle bacilli rarely ever hatch. Koch and Rabinowitsch found that 90 per cent of the embryos died. One chick hatched out but died of generalized tuberculosis in 75 days. It would appear therefore, that transfer of the infection through the egg to the succeeding generation rarely takes place.

The menace of avian tuberculosis to other animals is considerable. Mohler and Washburn of the United States Department of Agriculture in 1910 observed cases of avian tuberculosis in swine, as well as did European investigators before and since that time. The importance of this form of infection was not realized until after the nationwide program of eradicating bovine tuberculosis was started in 1917. Hogs from accredited areas where no bovine tuberculosis was supposed to exist were being received at slaughter houses in numbers nearly as large as previously. An investigation showed the disease was due to the avian type of organism. Observations carried on at the Illinois Experiment Station indicated that a single feeding of avian tubercle bacilli or of organs of a tuberculous bird was sufficient to produce the disease in hogs. Hogs receiving grain mixed with droppings of tuberculous chickens or allowed to associate with such chickens likewise developed the disease. A period of sixty days or less was required for the development of tuberculosis in hogs.

Calves are susceptible to the avian type of infection to a much less degree. At the Wyoming Agricultural Experiment Station (18) calves injected beneath the skin with living cultures of such organisms developed well marked local lesions. When injected intradermally, no disease developed. Removal of skin lesions by surgical operation resulted in subsequent negative tuberculin tests with no evidence of a recurrence of the infection. Graham found that calves associating with tuberculous poultry developed positive tuberculin reactions with local lesions. The avian type of disease is of comparatively small consequence in the bovine species.

The horse, ordinarily comparatively resistant to tuberculosis of all kinds, was found susceptible to the avian bacillus by Watson and Heath of the Canadian Department of Agriculture (19). The disease was quite rapidly fatal, developing a septicemia without tubercles. McFadyean describes a similar case of avian infection in a horse. The British Royal Commission on Tuberculosis induced tuberculosis in horses both by injection and by feeding avian baccilli. Bang produced a fatal tuberculosis by feeding a foal avian bacilli but old horses were not affected as evidenced by autopsy eight months later. Titze, however, fed a six-months-old foal forty-four culture tubes of avian bacilli, spread over a period of three months with no results. Calmette states that intravenous injection of horses with avian bacilli results in a generalized tuberculosis with more or less rapidity according to the dose.

Among the laboratory animals white mice and rabbits may be infected by ingestion or inoculation. Rabbits injected intravenously develop the Yersin type of disease without tubercles, dying rather quickly. Guinea pigs are ordinarily rather resistant to infection with the avian bacillus, although Hastings and Halpin and others have encountered strains which were quite virulent for such animals. Wild rats prove slightly susceptible to avian tuberculosis but no evidence presents itself to suggest that the disease appears among them spontaneously on premises where infected poultry is kept.

Avian tuberculosis in mammals other than hogs is of comparatively minor economic importance, with the possible exception of such animals acting as reservoirs of infection to re-introduce the disease in healthy poultry. This menace is likewise small compared to that of sparrows and pigeons acting as carriers from farm to farm. Graham (20) reports pigeons that were shot more than a mile from infected premises showing gross lesions of tuberculosis at autopsy. English sparrows and pigeons, found in the vicinity of infected flocks, were so far advanced with tuberculosis that they were unable to fly. There was no evidence that the disease was passed from pigeon to pigeon or sparrow to sparrow, the probability being that infection arose in the poultry yard.

The menace to man of avian tuberculosis is perhaps comparatively slight due to natural immunization of the human kind combined with a rather complete cooking of infected material. The literature, however, contains a record of a score or more of deaths of people infected with this type of organism. Lowenstein describes two cases of renal tuberculosis in children, apparently contracted from eating raw eggs. Jansco and Elfer found avian tubercle bacilli in the mesenteric glands of a small girl. Lipschutz frequently encountered avian organisms in cutaneous tuberculosis. Weber isolated them from the feces of a tuberculous patient. Max Koch and L. Rabinowitsch grew them from the spleen of a man dead of tuberculosis. Similar citations could be multiplied, while there have been, without doubt, numerous cases where no scientific study was made of the type of infecting organism. Eggs laid by fowls infected with avian tuberculosis are perhaps the chief source of danger to man. Under the ordinary methods of cooking, tubercle bacilli would not be killed but would be consumed in the living virulent state by whoever ate the eggs. The most common seat of the disease in fowls is the liver, which contains very characteristic yellowish white tubercles varying from a pin point to a hazelnut. Such organs unless completely cooked would be very dangerous to man. As a rule, however, the macroscopic appearance is such as to cause the organ to be discarded.

Tuberculosis in Swine

In 1908 when post-mortem inspection of meat animals in its present form was inaugurated by the Federal Government, less than four per cent of hogs were found infected with tuberculosis at Chicago. This figure increased until 1922 when 20.3 per cent hogs were retained for this disease. In 1917 the eradication program in cattle was undertaken with effective results from the cattle standpoint. The best information of the time led the packers to believe that 90 to 95 per cent of all tuberculosis in hogs was contracted from cattle. As a special encouragement to the area plan of tuberculosis eradication, the packers offered a premium of ten cents per live hundred weight on hogs marketed from the accredited area. This ten cent premium was a great stimulus in starting work under the area plan, but when the hogs from the first accredited areas came in as many animals were retained for tuberculosis as formerly. Extensive investigations showed that three-fourths of the hogs were suffering from tuberculosis of the avian type instead of the bovine type.

Graham and his co-workers at the University of Illinois studied 6,041 animals in 85 car lots. Of 62 animals in which the glands were typed, 82.2 per cent showed avian infection, 11.2 avian and mammalian mixed, while only 6.4 per cent were mammalian alone (21).

In the last few years the percentage of hogs retained for tuberculosis has fallen off somewhat (table 10). During the first half of 1926, 17.7 per cent of all hogs slaughtered were retained while 17.5 per cent of those from accredited areas were retained under the same inspection. The advantage was only 0.2 per cent in favor of accredited hogs.

Avian tuberculosis in hogs from the eastern states is much less prevalent than in the middlewest, because poultry is infected to a much less degree there.

Hogs may develop the bovine type of tuberculosis from drinking infected milk or from following infected cows in the barn yard. In England this type seems to predominate. Among 59 cases studied by the English Royal Commission, the bovine type was found 50 times, the avian 5 times, the human 3 times, with mixed bovine and avian once. Griffith and Eastwood had a similar experience, finding the bovine type 47 times, the avian 26, the human once, with 2 mixed and 2 atypical.

Hogs are quite susceptible to the human type of tuberculosis, which is the most serious of the three types of infection because of the danger of passing the infection back to man.

Recently Butler and Marsh (22) studied hogs fed on raw garbage from a tuberculosis hospital. Among 86 hogs slaughtered the human type of infection was found in 26, or over 30 per cent. This was confirmed by typing tests and inoculations. Disease of the glandular type developed in hogs within a period of 60 to 120 days after feeding began. When the organisms from these hogs were injected into laboratory animals the disease showed a marked tendency to localize in the lungs.

Tuberculosis of hogs is ordinarily of the glandular type. Since the life of such animals is usually brief, the disease develops no further. In older hogs lesions of the lungs and spleen may be

Table 10

SWINE RETAINED FOR TUBERCULOSIS, CHICAGO MARKET
1908–1927

Year	Hogs Slaughtered	Hogs Retained	Per cent Retained
1908	6,110,746	230,478	3.77
1909	6,172,124	258,224	4.18
1910	4,831,848	256,577	5.31
1911	5,394,544	408,709	7.58
1912	6,227,730	534,829	8.59
1913	5,843,182	566,779	9.70
1914	5,917,396	665,443	11.24
1915	6,548,442	816,020	12.46
1916	7,343,746	1,161,339	15.81
1917	7,550,530	1,229,297	16.28
1918	6,692,697	1,016,171	15.15
1919	8,359,895	1,104,631	13.21
1920	6,772,692	1,018,632	15.04
1921	6,311,332	1,117,307	17.70
1922	6,073,928	1,233,101	20.30
1923	7,524,553	1,433,648	19.04
1924	8,438,970	1,665,283	19.74
1925	7,159,623	1,351,612	18.88
1926	5,280,071	920,823	17.63
1927	5,655,764	910,463	16.09

found. The avian type is said to localize more strictly in the glands of the digestive tube than does the bovine.

PREVENTION AND CONTROL

The transmission of tuberculosis from animals to man is narrowed down to the food supply. Because the cow has closer contact with man by way of the milk supply than does either the hog or the chicken, bovine tuberculosis is relatively of more importance than the avian type.

The history of the control of bovine tuberculosis in man divides itself into several distinct periods. It began with the discovery by Robert Koch of the tubercle bacillus in 1882 and of tuberculin in 1890. While the value of tuberculin was being tested by many investigators, other researches demonstrated the presence of tubercle bacilli in milk. All this led to the controversial period of the early ninteen hundreds when Koch tried to prove that because cattle were not susceptible to human tubercle bacilli, man could not be infected with bovine strains. The fallacy of Koch's views was soon shown by many investigators.

The third period might be termed one of opposition. Along with the conclusive demonstration of the pathogenicity of bovine tubercle bacilli for man came other demonstrations not only of the large number of tuberculin positive dairy cattle but also new proofs of milk supplies infected with tubercle bacilli. Out of this grew certain antagonisms between health authorities on the one hand and dairymen on the other hand which can be well illustrated by a law passed by the Illinois State Legislature in 1911.

"An Act to prohibit the establishing and enforcing of the tuberculin test for dairy animals by any city, village, incorporated town, county or other corporate authority in the State of Illinois. Filed June 12, 1911, in force July 1, 1911.

SECTION 1. Be it enacted by the People of the State of Illinois, represented in the General Assembly: That it shall be unlawful for any city, village, incorporated town, county or other corporate authority in the State of Illinois by ordinance, rule or regulation other than may be established by the law of this State, to demand, fix, establish or require the tuberculin test to be applied to dairy animals as a means or measure of regulating and purifying milk, skimmed milk, cream and dairy products of said animals in any manner whatever, and every such ordinance, rule, by-law or regulation heretofore or hereafter passed, demanded fixed, established or required by any such authority other than the State of Illinois, is hereby declared to be void and of no effect."

The fourth period, that of eradication, began in 1917 when the Federal Government launched its accredited herd plan. So rapid have been the accomplishments in the last ten years, that Dr. J. A. Kiernan, Chief of the Tuberculosis Eradication Division of the United States Department of Agriculture has remarked, "Tuberculosis of livestock is losing its foothold in North America."

Three methods to prevent the transmission of bovine tuberculosis to man are in operation:

1. Eradication of tuberculosis in animals.
2. Careful supervision of the food supply through inspection of meat, and the pasteurization of milk, butter and cheese.
3. Immunization against tuberculosis.

Eradication of Tuberculosis in Animals

The tuberculosis eradication plan of the Federal Government begun in 1917 is threefold.

The accredited herd plan is a system of accrediting individual herds which pass two annual or three semi-annual tuberculin tests without reactors.

The area plan calls for the eradication of the disease within a circumscribed area such as a township or county.

The modified accredited area allows cattle from that area to be shipped interstate without a further tuberculin test when all cattle within the area have been tested and not more than 0.5 per cent have been found to react.

In 1918 only 22,212 cattle had passed one test; by 1927 more than thirty-nine million had been tested of which 1,250,000 reacted (table 11).

Tuberculosis in swine and poultry is more an economic problem than a public health problem. The number of human infections from these sources are relatively insignificant due to proper cooking of the pork and poultry before human consumption. Since swine are suceptible to the human type of organism, they are of potential danger in passing the disease back to man. Garbage or other food that might be contaminated with human tubercle bacilli should be cooked before being fed to swine. The bovine infection in swine can best be prevented by isolation from infected cattle and pasteurizing or boiling the milk fed to them. The eradication program in cattle is rapidly solving the problem of bovine infections in hogs. Avian tuberculosis in hogs can be eliminated only by the elimination of the disease in poultry. This can be accomplished through tuberculin testing of hens and breeding only from healthy stock. E. R. Smith recommends the practice of selling off every bird at the end of the first year of egg production. This would result in very little if any tuberculosis in poultry with the consequent reduction of the disease in hogs. The danger to man from infected eggs is not great but it still exists. Ordinary methods of cooking are not sufficient to destroy tubercle bacilli; elimination of the disease in poultry therefore is the only real safeguard.

Table 11

PROGRESS OF WORK OF ESTABLISHING ACCREDITED HERDS FREE OF TUBERCULOSIS

Fiscal Year	Cattle Tested	Number Reactors	Per cent Reactors	Accredited		Passed one test	
				Herds	Cattle	Herds	Cattle
1918	134,143	6,544	4.9	204	6,945	883	22,212
1919	329,878	13,528	4.1	782	19,021	6,535	117,243
1920	700,670	28,709	4.1	3,370	82,986	16,599	197,577
1921	1,366,358	53,768	3.9	8,201	193,620	49,814	643,233
1922	2,384,236	82,560	3.5	16,216	363,902	161,533	1,548,183
1923	3,460,849	113,844	3.3	28,526	615,156	312,281	2,724,497
1924	5,312,364	171,559	3.2	48,273	920,370	529,018	4,772,836
1925	7,000,028	214,491	3.1	72,383	1,275,063	921,758	8,047,540

Pasteurization

Pasteurization of milk was first practiced secretly by the dairy interests as an economic measure to improve the keeping quality of their product. Since otherwise filthy and unusable milk found its way to the market, health officials frowned upon the process. It was not long however, before its benefits were recognized and compulsory regulations passed for the pasteurization of all milk. Faulty equipment for many years raised some question as to the value of pasteurization, especially in regard to the tubercle bacillus. In recent years sanitary engineers have solved most of the perplexing problems and put the process upon a sound basis. Many cities and some states have passed specific regulations concerning the operation of pasteurization plants. In Illinois all such plants must obtain a license from the State Department of Public Health which calls for certain minimum requirements that insure the complete pasteurization of every drop of milk. A mobile laboratory installed in an automobile bus visits the plants to check up on efficiency and to help solve difficult problems. In this manner pasteurization has been made a complete safeguard against tuberculosis.

The efficacy of the pasteurization of milk even under the old methods where inaccuracies crept in from time to time is well demonstrated in New York City (33). The non-pulmonary death rate from tuberculosis per one hundred thousand in that city was 29 in 1910 and only 12 in 1925. In 1914 the pasteurization of milk, except certified, was made obligatory. In a very short time the non-pulmonary tuberculosis death rate started to fall with the above result. Examination of tuberculous glands of the neck in years previous to pasteurization revealed that in more than 50 per cent the process was due to bovine bacillus, whereas in only 6 of the 50 specimens obtained since pasteurization has become general was the bovine bacillus found and 5 out of these 6 cases were from out of town patients who had been fed on raw milk (table 12).

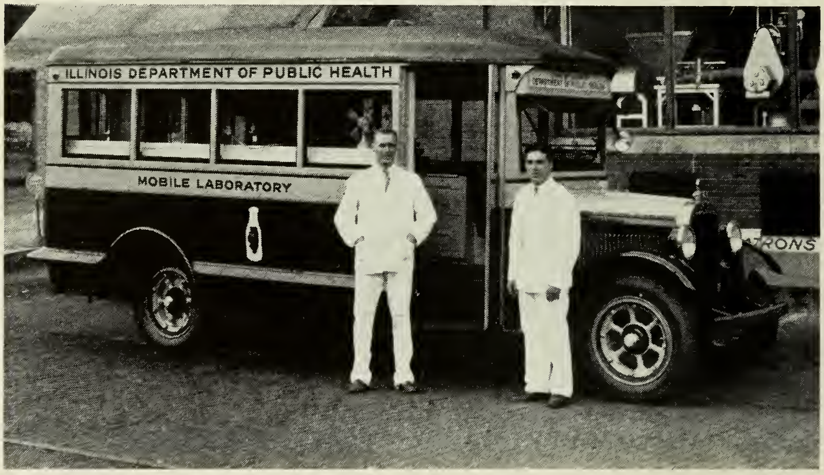


FIG. 3.—Mobile laboratory maintained by Illinois Department of Public Health. Frequent inspection of equipment of milk pasteurizing plants supplemented by laboratory examinations insures proper pasteurization of milk.

In New Haven 44 per cent of 41 raw milk samples were found to harbor tubercle bacilli in 1923; after pasteurization not a single sample was found to contain living tubercle organisms (23).

Table 12

PERCENTAGE OF BOVINE AND HUMAN TYPES OF TUBERCLE BACILLI IN TUBERCULOUS CERVICAL LYMPH NODES AND IN MENINGITIS IN NEW YORK CITY

Before and after the adoption of general pasteurization of the milk supply.

	Human	Bovine	Per cent bovine
1. Before the general pasteurization of milk but after the adoption of the general use of heated milk for infant feeding with the exception of certified milk.			
Tuberculous meningitis	54	3	5.3
Tuberculous adenitis (cervical)	34	21	38
2. After pasteurization of the general milk supply but with still the exception of certified milk. This protected raw milk amounted to about 1.5 per cent of the whole supply.			
Tuberculous meningitis	48	3*	5.8
Tuberculous adenitis (cervical)	30	6†	16

* There was a history in the case of one of the three, a child two years old, of having been fed on pasteurized milk. In the other two no clear history could be obtained.

† 2 cases; onset of disease in prepasteurization days.
2 cases; grade A raw milk and certified raw milk.
1 case; fed on ordinary raw milk.
1 case; seven years of age; according to history, was never given cow's milk. Butter might have been the source.

Winslow and Gray studied the tuberculosis mortality in numerous cities, with special reference to the pasteurization of the milk supplies. They conclude:

> "It would appear probably that the death rate from nonpulmonary tuberculosis of children between 1 and 4 years of age in cities which have a thoroughly pasteurized milk supply is about 75 per 100,000 population, as against a pulmonary rate of about 25 per 100,000. In cities which have not effectively pasteurized their milk supplies the pulmonary rate is about the same, but the nonpulmonary rate rises to about 100 per 100,000. An excess of 25 deaths per 100,000 indicates the approximate danger from bovine tuberculosis at this age period."(24)

Park (33) has recently studied the thermal death points of tubercle bacilli at different temperatures, showing that such organisms die in six minutes at 145°F. and in ten minutes at 142°F. The usual requirements of 142°F. to 145°F. for pasteurization give an ample margin of safety (table 13).

The pasteurization of all ordinary market milk for human food is a primary necessity. The tuberculin testing of cattle is of value as a means of controlling bovine infection for the good of the cattle industry. It may supplement pasteurization but it can not replace it as a means of protecting human lives.

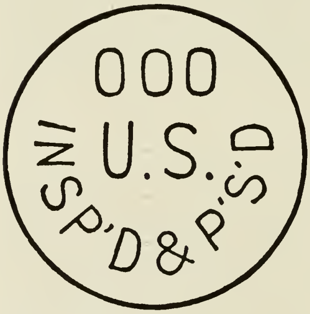
Table 13

THERMAL DEATH POINTS OF TUBERCLE BACILLI
(Park)

Temperature Fahrenheit	Time of exposure required to kill
170°	20 seconds
160°	30 “
155°	1 minute
150°	2 minutes
145°	6 “
142°	10 “
140°	15 “
138°	20 “
136°	30 “
134°	40 “
132°	60 “

Meat Inspection

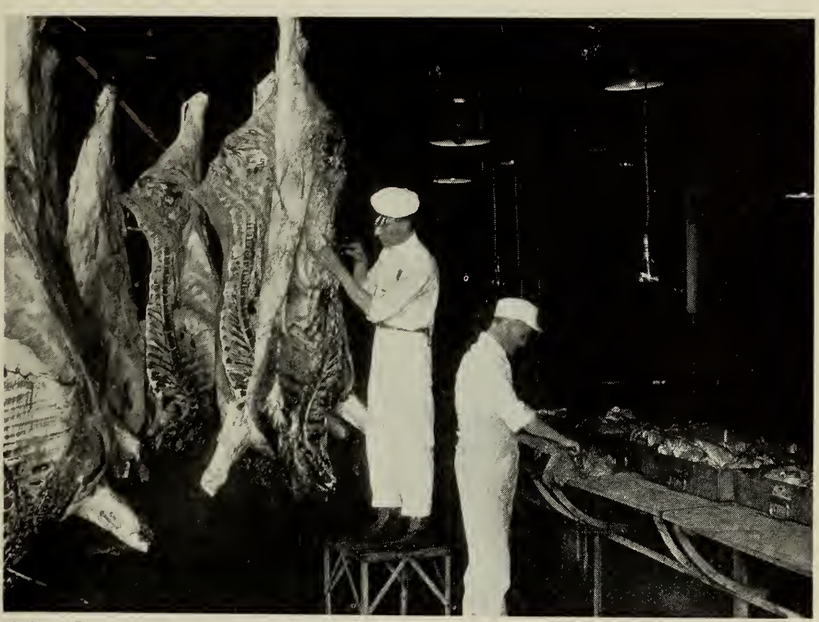
The protection of man against infection through meat is usually accomplished by rather complete cooking. In Germany according to Calmette, even meat which has been seized at the slaughter houses as unfit for sale, is cooked in large autoclaves and sold to the public at reduced prices. Infection of slaughter house workers has been noted by a number of observers in Europe and the United States. This is an industrial problem which can only be solved by the exercise of care on the part of individual workmen. Supervision by the United States Department of Agriculture eliminates from the market contaminated meat killed at the regularly established abattoirs producing meat for interstate shipment. Much meat finds its way to local markets, however, from farms where diseased meats do not go through any inspection. This is a potential source of danger.



United States Department of Agriculture

Fig. 4.—The "little purple stamp" used by inspectors of the United States Department of Agriculture for marking carcasses and products that have passed inspection. The three ciphers occupy the space where the number of the establishment appears.

Meat inspection is especially important in bovine tuberculosis. In 1926 out of 83,460 carcasses of cattle condemned in the United States, 53,512 were for this one cause alone (table 14). In 1891 the United States Department of Agriculture instituted inspection service at slaughter houses in six cities. While anti-mortem inspection was mandatory, post-mortem examination was discretionary. Wholesale meats were so marked, but there was no way to enforce the destruction of diseased carcasses, which could be sold if desired. There was no sanitary inspection of plants and no prohibition of harmful chemicals for adulteration or preservation. In 1906 the powers of the Secretary of Agriculture were greatly enlarged, and the appropriations for meat inspection made adequate. Inspections, which were limited to 163 establishments in 58 cities in 1906, were extened to 708 establishments in 186 cities in 1907. In 1927 there were 863 establishments in 258 cities and towns under Federal supervision.



United States Department of Agriculture

Fig. 5.—Federal inspectors making final examination of beef carcass.

Table 14

DISEASES AND CONDITIONS FOR WHICH CONDEMNATIONS WERE MADE ON POST-MORTEM INSPECTION—1926

Cause of Condemnation	Cattle		Calves		Sheep		Goats	Swine		Horses	
	Carcasses	Parts	Carcasses	Parts	Carcasses	Parts	Carcases	Carcasses	Parts	Carcasses	Parts
Actinomycosis	480	96,728	29	1,623		1					
Anthrax								13			
Asphyxia	4		6		10			1,115			
Blackleg	14		6								
Bone diseases	150	11	130	12	260	138		6,692	75		
Caseous lymphadenitis					1,999	9	5				
Cellulitis								22	847		
Congestion	4		1		16			20			
Contamination		248	4	3				738	2,327	1	
Cysticercus	169	1,359	26	4	172	2		71			
Dropsical diseases	27		3		37			84			
Emaciation	6,972		1,610		3,729		44	1,125		20	
Gangrene	62		27		7		1	32			
Hog cholera								42,337			
Hydronephrosis	1				1			30			
Icterus	93		152		1,123		1	4,438		1	
Immaturity			3,514								
Injuries, bruises, etc.	3,468	625	780	92	715	206	4	1,135	10,173	14	3
Leukemia	817		23		4			240		1	
Melanosis	32	16	49	4	22			96	3		
Moribund	9		2		39			107			
Necrobacillosis	6							1			
Necrosis	1	1,334			1	2		7	2		
Parasitic diseases	14	21	1		5			73			
Phlebitis			54								
Pneumonia, peritonitis enteritis, pleurisy, etc.	10,185		2,169		6,602		10	28,812		47	
Pregnancy and recent parturition	43							11		7	
Rabies	1										
Septicemia, pyemia, uremia, etc.	5,323		1,171		1,444		4	21,772		50	
Sexual odor					2		3	2,353			
Skin diseases	1		2		5			36			
Strangles										4	
Texas fever	90		148								
Tuberculosis	53,512	80,952	634	791				59,656	599,873		
Tumors and abscesses	1,982	4,221	68	542	221	33	2	2,575	256,633	23	16
TOTAL	83,460	185,515	10,609	3,072	16,414	391	74	173,591	869,933	168	19

Immunization Against Tuberculosis

For more than 40 years experiments have been carried on in the hope of finding a satisfactory method of immunizing against tuberculosis. These attempts fall into three groups—the use of living virulent tubercle bacilli, the use of living avirulent organisms and the use of dead organisms (25).

Living virulent tubercle bacilli are such a potential source of danger that their use can be discouraged in attempts at immunization since the danger is greater than any benefits which may be derived from the process. A further discussion of this method therefore need not be undertaken.

The use of dead tubercle bacilli for immunization purposes has been attempted by numerous workers with only partial results. Petroff claims that dead bacilli will protect an animal against an infecting dose but not a massive dose. Since a relative immunity rather than an absolute is perhaps all that can be hoped for, this method is one that possibly could be developed into considerable usefulness. At least it is safe.

Living avirulent organisms as proposed by Calmette and his coworkers in France are demanding considerable attention today. The so-called BCG organism (bacillus of Calmette and Guerin) was isolated in 1908 and has passed more than 230 successive subcultures on a special bile-gylcerin medium. The virulence of the organism has apparently been reduced by a lowered surface tension something after the manner of attentuation of virulent organisms proposed by Larson. Numerous investigations, on not only various species of animals, but also children have been carried on by Calmette.

The vaccine is administered to new born calves by a simple subcutaneous injection. There is no abscess or at most an insignificant one at the site of inoculation. The lesion which forms is microscopic in size, showing no central caseation and disappearing completely without scar formation. Abundant antibodies are produced in the body of the host, thus protecting against the invasion of virulent organisms. From 1921 to 1926 Calmette (27) vaccinated 3,051 calves with not a single accident. All animals were reported to remain well and healthy in spite of the fact that many of them were born of tuberculous mothers and were allowed to remain with them, drinking their milk.

In other countries the use of BCG has not been so encouraging. Schroeder of the United States Department of Agriculture failed with it. In Canada, Watson (26) found little difference in the resistance of cattle treated with BCG, treated with strains of low virulence or not treated at all (35). The Illinois Department of Agriculture in experiments culminating in 1928 had very unsatisfactory results with BCG. Larson injected the material into one series of calves, and another series of cows, repeating the administrations a month later. After an interval of six months these animals, together with an equal number of untreated controls, were turned in with a group of "spreaders"; contact was allowed to continue over a period of two years. The vaccinated cows showed no resistance to tuberculosis whatever while the calves so treated showed only slight protection.

One of the chief dangers with the use of BCG is that the avirulent organism which has established a living focus of infection within the body may at some later date resume its virulency. Recently Petroff isolated two types of organisms from BGG, one of which invariably infected guinea pigs (32).

In experiments carried out by the Illinois Department of Agriculture, Larson obtained very encouraging results with virulent tubercle exploded by carbon dioxide. A subcutaneous injection of such material repeated a month later by a second injection apparently protected most of the cattle under observation. The untreated controls were without exception infected while 11 out of 17 vaccinated animals were entirely free and 6 showed only slight lesions (13).

ITEMS OF NOTE

1. Tuberculosis whether in man or animals, is one of the penalties of civilization.
2. Man is moderately susceptible to the bovine type of infection.
3. Man is only slightly susceptible to the avian type of infection.
4. Milk under ordinary conditions of no control shows about 8 per cent of the samples infected with the bovine tubercle bacillus.
5. Proper pasteurization has been found an absolute safeguard.
6. Neither cattle nor poultry are responsible for the propagation of human tubercle bacilli.
7. Hogs are susceptible to all three strains of tubercle bacilli.

8. Immunization of calves by vaccination with living avirulent organisms has not given encouraging results in the United States and Canada.

BIBLIOGRAPHY

1. Calmette, Albert
   Tubercle Bacillus Infection and Tuberculosis in Man and Animals. Translated by Willard M. Soper and George H. Smith, 1923. Williams and Wilkins Co., Baltimore.
2. Bergey, David H.
   Manual of Determinative Bacteriology, 1925. Williams and Wilkins Co., Baltimore.
3. Rabinowitsch, L.
   Deutsch. Med. Wchschr., 1906, *32,* 866.
4. Nocard, E.
   Rev. gen. de med. veter., 1903, *1,* 1.
5. Cobbett, Louis
   Jour. State Med., 1922, *30,* 160-176.
6. Hoare, E. W.
   A System of Veterinary Medicine, 1913. Alex Eger, Chicago.
7. Douville, M.
   Rev. gen de med. veter., 1914, May 1.
8. Moore, V. A.
   Proceedings of the World's Dairy Congress, 1923. Vol. 2, P. 1482. Government Printing Office, Washington.
9. Kober, G. N.
   Am. Jour. Med. Sci., 1902, *126,* 684.
10. Park, W. H., and Krumwiede, Charles
    Jour. Med. Res., 1910, *23,* 205-368.
    Jour. Med. Res., 1911, *25,* 313.
11. Wang, Chung Yik
    Jour. Path. and Bact., 1917, *21,* 131.
12. Griffith, A. Stanley
    Report of Local Gov. Board. Public Health and Medical Subjects, 1914, *88,* 105-164.
13. Larson, W. P., and Evans, W. A.
    Jl. Am. Vet. Med. Asso., 1929, *74,* 581.
14. Gordon, J. Keith, and Brown, Elsie W.
    Am. Jour. Dis. Children, 1923, *25,* 234-246.
15. Tonney, F. O., White, J. L., and Danforth, T. F.
    Am. Jour. Public Health, 1927, *17,* 491-493.
16. Mohler, J. R., and Washburn, H. J.
    U. S. Dept. Agri., Bur. of Animal Indus., 25th Annual Report, 1908, p. 165-176.

17. FITCH, C. P., LUBBEHUSEN, R. E., and DIKMANS, RUTH N.
Jour. Am. Vet. Med. Asso., 1924, *66,* 43.

18. ELDER, CECIL, and LEE, A. M.
University of Wyoming, Agricultural Experiment Station, Bull. 140, May, 1925.

19. WATSON, E. A., and HEATH, L. M.
Am. Rev. Tuberc., 1925, *12,* 116-123.

20. GRAHAM, ROBERT and TUNNICLIFF, A. E.
University of Illinois, Agricultural College and Experiment Station, Circ. 285, July, 1924.

21. GRAHAM, ROBERT, TUNNICLIFF, A. E., and FRANK, E. R.
University of Illinois Agricultural Experiment Station Report, 1925. pp. 61-68.

22. BUTLER, W. J., and MARSH, HADLEIGH
Jour. Am. Vet. Med. Asso., 1927, *70,* 786-790.

23. BARTLETT, C.
Am. Jour. Public Health, 1923, *13,* 807.

24. Winslow, C.-E. A., and GRAY, CORA E.
Am. Rev. Tuberc., 1924, *10,* 186-202.

25. PETROFF, S. A.
Jour. Am. Med. Asso., 1927, *89,* 285-293.

26. WATSON, E. A.
Jour. Am. Vet. Med. Asso., 1927, *71,* 732-741.

27. CALMETTE, A., GUÉRIN, C., NEGRE, L., and BOQUET, A.
Ann. de l'Inst. Past., 1926, *40,* 89-133.

28. Same—1927, *41,* 201-232.

29. WEEKLY BULLETIN, City of New York, Department of Health.
1928, *17,* 93.

30. POIX, G.
Presse Med., 1928, *36,* 868-880.

31. WILLIAMS, R. S., and HOY, W. A.
Jour. Hyg., 1927, *27,* 37-39.

32. PETROFF, S. A.
Jour. Bact., 1928, *15,* 40.

33. PARK, W. H.
Am. Rev. Tuberc., 1927, *15,* 399-418.

34. RAVENEL, M. P.
Lancet, Aug. 10, 1901, pp. 349-356, 443-448.

35. WATSON, E. C., McINTOSH, C. W., and KONST, H.
Jour. Am. Vet. Med. Asso., 1928, *73,* 799-816.

CHAPTER II

ANTHRAX

Anthrax is primarily a disease of animals; secondarily man is infected from an animal either by direct or indirect means. Other designations for the trouble which have been used are malignant pustule, splenic fever, charbon and murrain.

HISTORY

The origin of anthrax is lost in antiquity. About 1491 B.C. Moses is quoted as threatening Pharaoh with an epidemic among his horses, cattle and sheep as follows:

> "Behold the hand of the Lord is upon thy cattle which is in the field, upon the horses, upon the asses, upon the camels, upon the oxen, and upon the sheep: there shall be a grievous murrain" (1).

The disease is considered by many authors to have been anthrax. The severity of the outbreak is indicated by the statement that all the cattle of Egypt died.

Classical writers in the centuries that followed allude to outbreaks among animals that in all probability were anthrax. Homer mentions it; Virgil describes it quite accurately in epidemic form in animals; Hippocrates, Galen and Pliny describe carbuncles which are considered anthrax by modern authorities. Numerous writers during the medieval ages mention devastating epidemics of the disease in animals. Not till near the end of the sixteenth century, however, was it suspected that the disease was transmissable to man. In 1613 in the south of Europe it developed into a scourge which carried off 60,000 people. In ancient Anglo-Saxon records there appear accounts of recipes, charms and incantations for the cure or prevention of "black bain" as anthrax was then called.

The beginning of scientific literature on anthrax is marked by the publication of "Charbon Malin" by Fournier in Dijon, France, in 1769. The Academy of Dijon offered a prize for essays on the subject which resulted in some remarkably accurate descriptions of the disease in man and animals. Chabert (2) in 1780 distinguished different forms of anthrax in man from other septic conditions of the skin, and Barthelemy in 1823 proved the contagious nature of the disease in animals.

The cause of anthrax at this time was a muted point, theories being bound up in all the hazy and illogical notions of the pre-Pasteur period. Delafond (3), a French veterinarian, held that anthrax in sheep was caused by an "excess of blood circulating in the vessels due to too copious and substantial feeding." Heusinger (4) was of the opinion that anthrax in man was due to a malarial neurosis dependent upon various conditions of the soil and climate. Other ideas included the influence of soil, the summer heat, storms, insanitary conditions of the stables and the like.

Among such a confusion of ideas, with anthrax killing off from 25 to 50 per cent of the flocks of sheep and so prevalent in France and other European countries that farming was almost at a standstill (5), a period of observation and investigation began about 1850 which culminated in the identification of the anthrax bacillus by Koch in 1876 and the production of a preventive vaccine by Pasteur in 1880.

Historically anthrax has a double significance. It was the first disease of man and animals shown to be caused by a micro-organism and the first disease against which a bacterial vaccine was found to be effective.

The Anthrax Bacillus

Anthrax bacilli in the blood were first seen by Pollender (6) in 1849. His description was not published until 1855, however, and meanwhile Rayer and Davaine had called attention to these peculiar bodies "about twice the size of a red blood corpuscle" which were present in the blood of infected animals. Brauell (7) in 1857 and Delafond in 1860 both observed the same organisms but none of the investigators up to this time realized their full significance. The researches of Pasteur into the field of what was destined to become the science of bacteriology stimulated Devaine (8) again to take up the study of these organisms. In 1873 he demonstrated that only the blood of animals sick with the disease contained the organisms and that when this blood was passed through a clay filter, it was harmless for other animals. In 1876 Robert Koch (9) cleared up the whole matter by cultivating the organisms outside the body in pure culture and again producing the disease.

Bacillus anthracis is one of the largest organisms among the pathogenic bacteria, measuring from 1 to 1.25 microns in thickness and 5 to 10 microns in length. It has the further distinction of being one of the most resistant of organisms among the pathogens. In its spore stage it has been found to survive in a dried condition for seventeen years (Aiello and Drago) and eighteen and a half years (Busson). Chemical disinfectants are not very effective against the spores except in strong solutions or over long periods of time—lysol 5 per cent seven hours, carbolic acid 5 per cent 2 days, mercury bichloride 10 per cent twenty minutes, formalin 10 to 20 per cent ten minutes, iodine 1 per cent 2 hours (20). In dry air they are destroyed at 140°C. in three hours or at 100°C. in twenty-four hours (31), in boiling water two hours may be required to kill them. The vegetative forms, free from spores, are easily destroyed by the usual methods of disinfection. Anthrax bacilli are easily cultivated on all ordinary nutrient media, forming characteristic colonies which it is not difficult to identify.

Geographical Prevalence

Anthrax occurs in all parts of the world. In certain European countries where restrictive measures have been enforced, together with preventive vaccination, as in England, France and Germany, the disease in animals is now quite rare. In other parts of the world, especially the Asiatic countries of Siberia, Persia, Tibet, China, India and Arabia, where sanitary science has not been developed, anthrax in animals is very prevalent. It occurs in many parts of South America, Africa and Australia.

Anthrax was brought to North America quite early in the days of the colonies. Carpenter (14) traced the infection in Louisiana back to the time of its settlement by the French. It was believed to have been prevalent among the deer of that region. The cattle were likewise attacked annually so that by 1835 it existed in all parts of the state. Carpenter states, however, that it "seems not to have been even noticed in the medical annals of America." In 1835 he reported a large number of human cases. Later references in that state as well as other lower Mississippi and Gulf States in the years that followed indicate that anthrax was more or less endemic in that region (12). The first human cases in the United States of which there is record, occurred in Philadelphia in 1834 (13). "Murrain," or anthrax, was at that time prevalent among cattle fed on the common. Several persons who skinned such animals developed lesions on the hands.

In New England, Osborn (11) could find no mention of anthrax in the colonial records of Massachusetts. In 1852 a report appeared by a physician in Salem, giving accounts of six different cases which he had seen in previous years (12). From 1853 to 1869 at Walpole, Massachusetts, 26 cases occurred traceable to a hair factory (11).

Numerous references have appeared since that time in the medical literature indicating that anthrax has existed especially in the northern states of the Atlantic seaboard and in the central Gulf states. In Louisiana in 1924, 20,000 head of cattle perished from the disease in 43 out of 64 parishes (15).

The Disease in Animals

Practically all animals, domestic and wild, are subject to anthrax, to a greater or less degree. Sheep and cattle are the chief sufferers, but other domestic animals such as horses, goats and even hogs may contract the disease on occasion. Dogs have been known to become infected through eating carcasses of animals dead of the disease. Fowls are practically immune except under adverse conditions such as those in Pasteur's classical experiment where the body temperature was lowered several degrees by partial immersion of the bird in cold water. The small laboratory animals such as guinea pigs, mice and rabbits die very quickly after inoculation.

Infection of animals ordinarily takes place by ingestion of the organisms rather than by contact. Pastures in which infected cattle have roamed may remain contaminated for years and prove fatal to any stock turned thereon to graze. Pastures contaminated by tannery wastes have been described by Eichhorn (16) and by others as a menace to cattle, as are streams into which tannery wastes are discharged and from which cattle or other animals may drink. Anthrax organisms may be carried from place to place by birds and small animals. They have been found on the beak and feet of buzzards which have fed on animals dead of the disease. The dog, cat, opposum and chicken all may harbor live spores in the feces after eating infected flesh and thus serve as dangerous carriers in spreading the contagion to new territory. Morris (17) cites a case in which it was claimed that virulent spores were found in the feces of a dog eight years later. Another method in which infection by ingestion takes place is through hay, forage or other crops which have been grown on infected ground or otherwise contaminated with anthrax spores. Smyth (20) reports a small epidemic in cattle in Maryland due to feeding infected blood meal.

Biting flies may be a factor in the spread of anthrax not only from animal to animal, but also from animal to man. According to Castellani anthrax spores will remain alive in flies for twenty days, being found in the feces. Infection through abrasions, cuts and scratches can likewise spread the disease, but such factors are of small importance in animals compared to ingestion.

The disease in animals may appear in three forms—apoplectic, acute or subacute. Washburn (18) described these different conditions as follows:

> "The apoplectic form is most frequently seen attacking cattle or sheep at the beginning of an outbreak before the animals of the vicinity have developed any degree of natural immunity to the infection. Here the animals present symptoms of cerebral apoplexy. They reel and fall, bloody liquid flows from the body openings, and death soon follows. If the body is opened and search is made for evidence of disease, it may be quite impossible to detect any definite lesions or change in the tissues.
>
> "The acute form of the disease develops more slowly, but becomes well established in twelve to twenty-four hours after the premonitory symptoms are noticed. In these cases the fever is intense (104° to 107° F.) The animal is greatly prostrated. Cerebral congestion causes excitement, which is followed by drowsiness and staggering gait. There is frequent passage of bloody urine, followed by convulsions and death. In this type of the disease, as well as in the apoplectic form, post mortem examination of the carcass may fail to reveal any definite lesions.
>
> "The third form of anthrax, the subacute, is the most common. The symptoms are like those of the acute form except that they are of slower development. Instead of becoming established in twelve to twenty-four hours, one to seven days may be required. The fever is very high. Serious colics are often present. Local anthrax tumors appear externally, first near the shoulders, neck and head, and are usually due to local injury or bruising, which gives rise to a collection of bacilli within the blood vessels of the part, whose resulting inflammation gives rise to the swellings or carbuncles. These tumors are at first hard and circumscribed, but later become cold, insensible, diffuse, and fluctuating. An examination of the carcass of an animal dead of anthrax of the subacute form will probably show many lesions or alterations. Hemorrhages may be found in almost all parts of the body. Serous infiltrations may be present beneath mucous membranes and skin. There will be swelling of the spleen, the liver and the kidneys, and the blood will be of a muddy or tarry appearance and incoagulable. The cavities of the body contain more or less bloody effusion,

and the lymphatic glands are swollen and contain small hemorrhages. The red blood cells have become broken down in large numbers and the serum of the blood has been markedly reddened. The walls of the intestines may appear perfectly normal, but hemorrhages are frequently seen, especially in the walls of the duodenum.

"The subacute form is the one most commonly met, and it is the only form which responds favorably to treatment. Death ensues so quickly in the other two forms that attempts at treatment are of but little use.

"Isolated or sporadic cases are usually of the subacute form, and are frequently limited to the formation of a tumor or carbuncle at the point of the body at which the infective germs first gained their entrance."

ANTHRAX IN MAN

The number of human cases of anthrax in the United States is not large. Figures gathered from various state health departments by Smyth (20) would indicate that there are usually not more than a hundred cases annually (table 15).

Table 15

HUMAN CASES AND DEATHS OF ANTHRAX OCCURRING IN MAN IN THE UNITED STATES

Year	No. of States reporting	Cases	Deaths	Percent Fatal
1919	7	81	23	28
1920	8	99	24	24
1921	7	70	24	34
1922	6	86	20	23
1923	8	65	11	17
1924	18	107	36	33
1925	15	106	11	10
1926	12	91	10	11

Andrews (12) compiled the records of 222 fatal cases occurring in the registration area of the United States from 1910 to 1917. He estimated that for each death there occurred four other cases. This figure is substantially in accord with that of the British authorities who placed the mortality at about 25 per cent in Europe (19). The number of human cases for the eight years included in Andrews study therefore would be about 140 annually.

Anthrax in man is usually a cutaneous disease. The organisms will not pass through the unbroken skin, but a slight abrasion or wound will allow them to enter. At the site of the wound there developes either a carbuncle (malignant pustule) or a malignant oedema. Sooner or later such lesions may lead to an infection of the whole body with death as a possibility. Such abrasions are usually situated on the head, neck and upper extremities (table 16).

Table 16

LOCATION OF LESIONS IN 114 CASES OF CUTANEOUS ANTHRAX IN MASSACHUSETTS (11)

Neck	45
Chin	8
Forehead	5
Eyelid	2
Cheek	17
Nose	2
Upper arm	2
Forearm	21
Hand	5
Back	1
Upper leg	2
Ankle	2
Unknown	2
	114

Table 17

CASES OF ANTHRAX BY OCCUPATION
Cited by Andrews (12)

Occupation	New York State	Philadelphia	Massachusetts	Total
Hide, skin, leather hair and wool industries	10	27	21	58
Transportation workers	3	—	9	12
Farmers	3	—	—	3
Veterinarians	1	—	—	1
Longshoremen	—	2	—	2
Miscellaneous	6	3	5	14
	23	32	35	90

Infection by inhalation of the dried spores occurs in dusty working establishments and results in a pulmonary form of the disease (wool sorters disease). While this mode of infection is comparatively rare, the mortality is quite high when it does occur.

Gastro-intestinal anthrax is contracted by ingestion of improperly cooked meat from infected animals. Infection by ingestion is the least common mode in man as opposed to the most common in animals.

Anthrax in man is practically always derived from an animal. There are a few cases on record where it has been passed from one person to another but they are comparatively rare. Bites of bloodsucking insects may at times be a factor, but such cases often can be traced to an infected animal rather than an infected person upon whom the insect has previously fed.

Bites from pets which have recently fed upon the carcasses of animals dead of anthrax have been known to cause anthrax infection. This again is of animal origin.

Contact with the animal may be quite direct, as a farmer caring for a sick animal or skinning one which has died from the disease. It may be more remote as handling hides which have been shipped long distances. Cases are recorded of workmen in tanneries carrying the infection home to members of their families, though escaping themselves. The contact may be very remote, such as a man living in a hotel in a large city and rarely even seeing an animal, becoming infected by a shaving brush made from the hair of an infected animal in China.

Anthrax an Industrial Hazard

Human anthrax in the United States is primarily an industrial hazard. Workers in the hide, skin, leather, hair and wool industries show a greater number of cases than in other occupations, followed by transportation workers, farmers and veterinarians. In Table 18 are the combined figures of instances cited by Andrews showing the occupations of cases of anthrax reported to the New York State Department of Labor from September, 1911, to March, 1916, of cases entering a contagious disease hospital in Philadelphia from January, 1909 to April, 1916, and of cases entering a hospital in Massachusetts from 1881 to 1916.

Table 18

DEATHS FROM ANTHRAX IN U. S. REGISTRATION AREA BY OCCUPATION, 1910-1917

Hide, skin and tannery works	42
Farmers, ranchmen or women living on farms	42
Transportation employees	25
Wool, hair and bristle workers or brush salesmen	19
Rag handlers in paper mills	2
Liverymen	2
Veterinarian	1
Laborers connected with above industries	10
Connection between occupation and infection obscure or non-existent	73

The total number of deaths from anthrax in the registration area in the United States from January, 1910 to December, 1917, tabulated by Andrews as to occupation, showed that 143 or nearly two-thirds of the persons under consideration were in occupations where the menace from anthrax was well recognized (table 18).

Smyth has collected figures on the sources of human anthrax infection in the United States, showing results similar to those obtained by Andrews.

A study of the 724 cases occurring between 1919 and 1927 bears out his statement that "the question of the danger of anthrax infection in industry from the handling of hides, skins, wool and hair is one of increasing importance in this country" (20).

Table 19

Cases of anthrax by occupation (Smyth)

	1919-23	1924-27	Total
Tanning Industry	120	68	188
Wool Industry	8	42	50
Cattle	32	17	49
Shaving Brushes	44	4	48
Hair and Fur	30	11	41
Veterinarians and Students	—	3	3
Transportation	—	2	2
Sources not stated	74	169	343
	409	316	724

In 1926 four states in which the tanning industry is largely centered contributed 59 cases of the total 91 reported from twenty-two states in the country, as follows: Pennsylvania 20, New York 13, New Jersey 13, and Massachusetts 13.

In Pennsylvania over a twelve-year period Smyth and Bricker (21) found that among 13,339 men working in tanneries, 1,040 were directly exposed to infection, and of this latter number, 123 cases of the disease developed, a rate of about 11 per cent.

Anthrax from Shaving Brushes

During the World War and in the years that followed there was much excitement over anthrax from shaving brushes, both in this country and in England. The source of nearly all the hair for shaving brushes was horsehair from Siberia and China, where anthrax was quite prevalent (22). During the course of shipment in normal times the material was cleaned and disinfected usually in France or Germany. With the advent of the

World War, shipments were made directly to the United States across the Pacific. Faulty or careless processes for insuring protection against the disease were responsible for a considerable number of cases.

The first recorded case of anthrax from a shaving brush was on July 9, 1915, in a British soldier (23). From that time on similar cases occurred not infrequently in both the British and American forces as well as the civil population. In the United States troops there occurred during the war 149 cases of anthrax with 22 deaths, due to shaving brushes. In New York City from 1919 to 1923 there were 32 cases reported from this source. In Illinois I examined one lot of three dozen brushes every one of which was contaminated with virulent anthrax spores. The United States Public Health Service together with the Department of Agriculture passed stringent regulations concerning the importation of hair and hides which afforded some protection. Several of the manufacturers of better grade brushes exposed their hair to special processes and labeled the brushes accordingly. Cheap brushes were usually most dangerous. With the advent of normal trade and manufacture, anthrax from shaving brushes has almost ceased to occur.

Prevention and Control

The prevention and control of anthrax in man depends upon three factors—eradication of the disease in animals, elimination of industrial infections, and finally, better use of anti-anthrax serum for the cure of infected cases.

The eradication of anthrax in animals is a difficult task, but is being accomplished as an economic measure in the animal industry. Vaccination of sheep, horses and cattle in infected areas is the most useful measure known. Such vaccinations do not afford protection for more than a year and must be repeated.

Pasteur in his early work used for a vaccine live anthrax organisms attenuated by growing at high temperatures. In 1902 Sobernheim used living virulent anthrax bacilli, at the same time giving an injection of anti-anthrax serum. Eichhorn (24) in the United States has prepared a spore vaccine of Pasteur's No. 2 strain (attenuated to kill a guinea pig but not a rabbit) which is used together with a potent anti-anthrax serum. Kelser (25) believes that in heavily infected districts a greater immunity is required than that obtained with the Pasteur No. 2 strain and recommends a more virulent strain.

Fields once infected with anthrax bacilli are difficult to cleanse. These should be turned to other purposes than grazing wherever possible. Earthworms which were once thought to be a factor in carrying anthrax spores from the depths of the soil to the surface are probably not a factor in the spread of the contagion. Animals dead of the disease should not be carried to new localities, but should be disposed of on the premises. They should either be burned or buried deeply with quick lime, without opening the carcasses or removing the skins. In the absence of oxygen, spores are not formed and the vegetative forms of the organism are easily destroyed. When the skin is opened, spores form with the access to oxygen, and the problem of destruction is greatly increased.

Many devices have been suggested for the sterilization of hides, none of which have been entirely satisfactory. Two considerations are of prime importance—economy of cost which will make the process come within the possibility of commercial use, and lack of injury to the hides and skin. Horse hair and wool can be sterilized quite satisfactorily, as is done at central disinfecting stations in England. It is said that the rather marked increase in anthrax from wool in the United States during the past three years is due to compulsory sterilization in England of wool coming from infected regions (20). Such material naturally finds its way to this country where the regulations are less strict. Smyth (32) has shown that iodine 1 per cent or iodine vapor will completely disinfect hides without injury but the cost is at present prohibitive.

The present government regulations aiming to prevent the introduction of anthrax on imported hides, hair and wool apparently are inadequate to accomplish the desired purpose. The elimination of this source of contagion would greatly reduce the cases of human anthrax in this country.

Anthrax as an industrial hazard requires the promulgation of educational measures among those most exposed to infection, better devices for the sterilization of hair and hides, and better government regulations to prevent the importation of infected hair and hides.

Serum for the cure of anthrax in man and animals has been in use since 1880 when Toussaint (26) first found that blood from animals dead of the disease, when heated and injected into other animals, would protect them against infection. In 1895 Marchoux (27) in France prepared an efficacious anti-anthrax serum which would cure sick animals. In 1903 Sclavo in Italy produced a serum that has been widely used for the cure of anthrax in man. Animals are more difficult to cure, due to the more acute course of the disease, and the use of serum is not prevalent. It has some value, however, and is worth trying when an attempt at cure of an animal is undertaken.

The value of anti-anthrax serum for the cure of disease in man has been demonstrated numerous times. Sclavo in his early work compiled statistics showing a mortality of only 6.09 per cent in 164 patients receiving serum against a mortality of 24.16 per cent in patients receiving no serum. Regan (28) quotes figures from the Argentine Republic where serum in 130 cases reduced the death rate to zero, the few fatal cases occurring being due to secondary infections.

In a report of the British Ministry of Health (29) on 800 cases, the mortality was only 4.0 per cent where serum alone was used against 48 per cent with no treatment.

Symmers (30) in 1920 treated fifteen patients with Eichhorn's serum in Bellevue Hospital, New York City without a death, whereas the mortality from anthrax for the five previous years was in that city 47 per cent. Regan treated eight cases with the same serum with complete recovery of all. Other investigators have reported many more cases treated with serum, mostly with favorable results.

Items of Note

1. Anthrax is primarily a disease of animals.
2. Practically all human infections arise from contact with animals direct or remote.
3. Anthrax in man in the United States is an industrial hazard at the present time.
4. The anthrax bacillus in its spore stage is the most resistant of all pathogenic bacteria.
5. The control of anthrax in animals depends upon destruction of infected animals, with vaccination of healthly animals in endemic areas.
6. The control of anthrax in man depends upon the promulgation of educational measures among industrial workers ex-

posed to infection, together with better devices for sterilization of infected hair, hides and wool.

7. The shaving brush has ceased to be a hazard.

BIBLIOGRAPHY

1. The Bible—Exodus 9:3.
2. Chabert, P.
   Description and treatment of Charbon in animals. Paris, 1780.
3. Delafond
   Cited by J. B Andrews. Bur. of Labor Statistics Bull. 267.
4. Heusinger, C. F.
   Anthrax: The Disease in Man and Animals. 1850.
5. Vallery-Radot, R.
   The Life of Pasteur. Doubleday Page and Co., New York, 1916.
6. Pollender
   Vierteljschr. f. gerichtl. Med., 1855, *8*, 103.
7. Brauell
   Virchow's Archiv. 1857, *11,* 132.
8. Devaine, C.
   Rev. Phot. d. hôp. de rend. Acad. de Sci. Paris, 1873, *5,* 221.
9. Koch, Robert
   Beitr. z. Biol. d. Pflanzen., 1876, *2,* 303.
10. Devaine and Rayer
    Comptes rendus de la Soc. de Biol., 1850, p. 141.
11. Osborn, Stanley H.
    Am. Jour. Public Health, 1920, *10,* 657-665.
12. Andrews, J. B.
    United States Department of Labor, Bur. of Labor Statistics, Bull. No. 267, 1920.
13. Pennock, C. W.
    Am. Jour. Med. Sciences, 1836, *19,* 13-25.
14. Carpenter, W. M.
    Southern Medical Journal, Feb., 1839, pp. 257-274.
15. News Note.
    Jour. Am. Med. Asso., 1924, *83,* 538.
16. Eichhorn, A.
    Am. Med. Vet. Asso. Jour., 1920, *58,* 278-288.
17. Morris, Harry
    Am. Med. Vet. Asso. Jour., 1920, *56,* 606-608.
18. Washburn, H. J.
    Farmer's Bulletin, 439, 1911, U. S. Dept. of Agriculture.
19. Bell and Legge
    In Allbutt and Rolleston's "System of Medicine," 1906, p. 252. Cited by Andrews, U. S. Dept. of Labor, Bur. of Labor Statistics, Bull. 267, p. 100.

20. Smyth, Henry Field
Am. Jour. Public Health, 1924, *14,* 920-924.
Also report of Anthrax Committee of American Public Health Association, Cincinnati, Oct. 20, 1927.
21. Smyth, H. F. and Bricker, E.
Jour. Ind. Hyg., 1922, *4,* 53.
22. United States Public Health Reports, 1919, *34,* 994-995.
23. Elworthy, R. R.
Lancet, 1916, *1,* 20-23.
24. Eichhorn, A.
United States Department of Agriculture, Bur. of Animal Ind., Bull. 340, 1915.
25. Kelser, Raymond A.
Manual of Veterinary Bacteriology, p. 211. Williams & Wilkins Co., Baltimore.
26. Toussaint, H.
Bull. Acad. de Med., Paris, 1880, *9,* 792.
27. Marchoux, E.
An. de l' Inst. Past., 1895, *9,* 785.
28. Reegan, J. C.
Jour. Am. Med. Asso., 1921, *77,* 1944-1948.
29. Mitchell, William
Brit. Med. Jour., 1911, *1,* 751-752.
30. Weekly Bulletin, New York City Health Dept., Aug. 7, 1920.
31. Smyth, H. F.
Am. Jour. Hyg., 1921, *1,* 541-555.
32. Smyth, H. F.
Am. Jour. Hyg., 1923, *3,* 224-237.

CHAPTER III

FOOT-AND-MOUTH DISEASE

Foot-and-mouth disease is an acute febrile affection caused by a filterable virus. During the course of the disease characteristic vesicles develop on the mucous membranes of the mouth and on the skin of the interdigital spaces.

Primarily it is a disease of cattle and other cloven-footed animals; secondarily other animals as well as man may become infected.

Other names under which the disease has been known are aphthous fever, epi~~c~~zoötic aphtha, infectious aphtha and eczema contagiosa.

HISTORY

The history of foot-and-mouth disease seems to be shrouded in uncertainty. For some centuries it has been known in Central Europe, but it was ascribed to numerous causes, such as climatic or meteorological conditions, bad air, excessive fatigue, etc. Its infectious nature was recognized in 1764, but until the development of bacteriology a century later, the etiological factor or the mode of spread was not known. Epidemics have recurred at frequent intervals in different parts of the world. As an illustration of its severity in Central Europe in 1911, Mohler relates that 3,366,369 cattle, 1,602,927 sheep, 2,555,731 hogs and 53,674 goats were affected, or approximately one out of every seven animals in that region.

In the United States foot-and-mouth disease has appeared in epidemic form among cattle on eight different occasions (2). In 1870 an outbreak, introduced from Scotland by way of Canada, spread into New England and New York state, but seems to have been arrested in a short time. In 1880 the infection was discovered in two or three lots of imported cattle, but the contagion was arrested before it could spread to other herds. In 1884 the third outbreak appeared at Portland, Maine, but was soon under control. All three of these epidemics were brought about by the importation of diseased cattle. Since then the United States Department of Agriculture has been established, and rigid inspection measures on cattle entering this country have been enforced.

In 1902 the disease appeared at Chelsea, Massachusetts, and again spread in New England. Mohler ascribes this outbreak to cowpox virus brought in from Japan for the production of smallpox vaccine. Cattle to the number of 4,712 were infected, while several hundred hogs, sheep and goats were slaughtered. In November, 1908, the next outbreak occurred in Michigan and spread to New York, Pennsylvania and Maryland. One hundred and fifty-seven premises were found infected and 3,636 animals slaughtered, valued at $90,033. Rosenau and Mohler showed conclusively that this time the disease was brought in through contaminated vaccine virus from Japan. The vaccine virus was used for the production of vaccine in calves. The calves were thus infected as well as the smallpox vaccine produced and distributed. A second firm purchased some of the contaminated vaccine and infected its own calves. The calves of the second firm were sold in the open market and started the epidemic. The Government immediately recalled all the contaminated vaccine distributed, and there is no record to show a human case resulted from its use. Since then the Federal Government has assumed rigid control of vaccines and other biological products, the United States Public Health Service being responsible for products intended for human use and the Bureau of Animal Industry those for veterinary use.

The most extensive invasion of hoof and mouth disease yet recorded in this country broke out in October, 1914, near Niles, Michigan. The origin is uncertain but it may have found its way in from South America with merchandise. The Chicago Stock Yards became infected with disastrous results. To complicate matters still further the National Dairy Show was on at the time. One hog cholera virus plant became infected also, and accidently distributed one lot of contaminated hog cholera virus. Altogether, 22 states and the District of Columbia were affected, mostly from Iowa to Massachusetts and 3,556 herds were slaughtered, valued at nearly $6,000,000.

The next appearance of the disease took place in 1924 in California. A picturesque incident that quickly caught the popular attention was the "border war" instituted by Arizona against introduction of the infection into that state. As a result of rigid quarantine regulations automobiles were prohibited from passing into Arizona from California. Cars and tourists accumulated at the border for several days until at one point more than 200 cars carrying 700 people were waiting. Finally they were allowed to go on their way after thorough disinfection.

The original infection in this epidemic was supposed to have come on a transport from the Orient with garbage used to feed hogs in California. From its focus around San Francisco bay the disease spread to sixteen counties in California within a period of six months, requiring the destruction of more than 58,791 head of cattle.

In September, 1924, the disease appeared in Texas but was brought under control within 30 days by immediate and complete slaughter of 148 herds. The source of the trouble is not known certainly, except that it probably had no relation to the California outbreak. It is thought that possibly it was brought in by members of crews from ships taking on cattle from infected foreign ports for food purposes. The next year a recurrence appeared which was immediately suppressed with the slaughter of 153 infected herds and 848 exposed herds.

Geographical Prevalence

Foot-and-mouth disease has prevailed at different times in nearly all parts of the world in all climates. In Europe it has become endemic, breaking out from time to time with tremendous losses, especially in France, Germany, Switzerland, Italy and other central countries. The isolation of Great Britain, Denmark, Norway and Sweden have made possible a fairly successful control. In Asia there have come reports from China, Russia, Japan and the Philippine Islands. In South America it has appeared in years past in Brazil, Argentine and Uruguay.

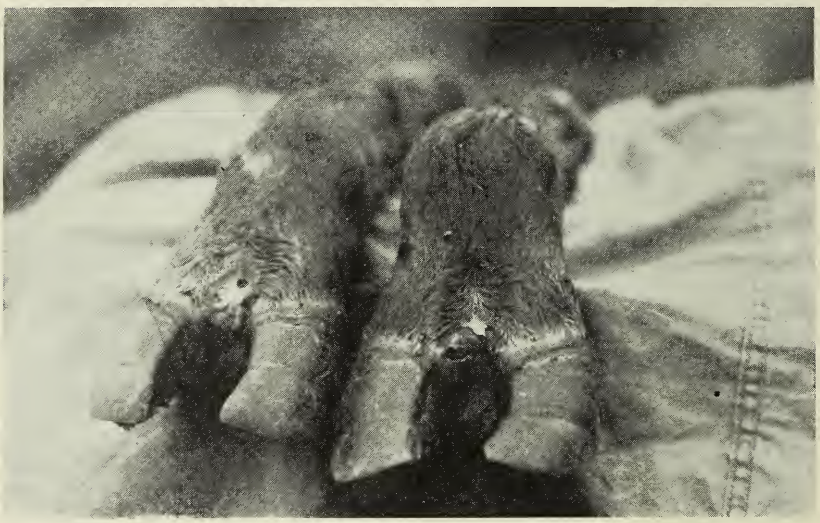
In the United States the eight outbreaks reported above have occurred from Maine to California. In each instance they have been quickly suppressed, however, so that the country may be said to be free of infection. The same is true of Canada and Mexico.

Animals Susceptible

Cattle, hogs, sheep and goats are very susceptible to infection, while the buffalo, American bison, camel, chamois, llama, giraffe and antelope follow closely in order, along with other cloven-footed animals.

Dogs, cats and poultry show slight susceptibility, but may become infected on occasion. The Berlin Commission found the dog and cat refractory; Olitsky found the horse refractory.

Among the laboratory animals the guinea pig is the animal of choice for experimental purposes. Some of the early investigators found guinea pigs refractory to infection and Hobmaier could pass the virus through them but a limited number of times before losing it. It is now generally accepted that the guinea pig is quite susceptible to infection with the virus and once the disease becomes established in this animal it can be propagated indefinitely by passage from animal to animal. Olitsky and Boez (3) passed the virus through 261 successive guinea pigs with no loss of virulence by injecting it intradermally into the foot pad. The disease progresses rapidly in such animals but does not spread naturally from animal to animal.



United States Department of Agriculture

FIG. 6.—Lesions on hoofs in foot-and-mouth disease.

Rabbits and rats on the other hand, are much less susceptible to infection than guinea pigs, and the disease following successful inoculation is correspondingly less marked.

The Infectious Agent

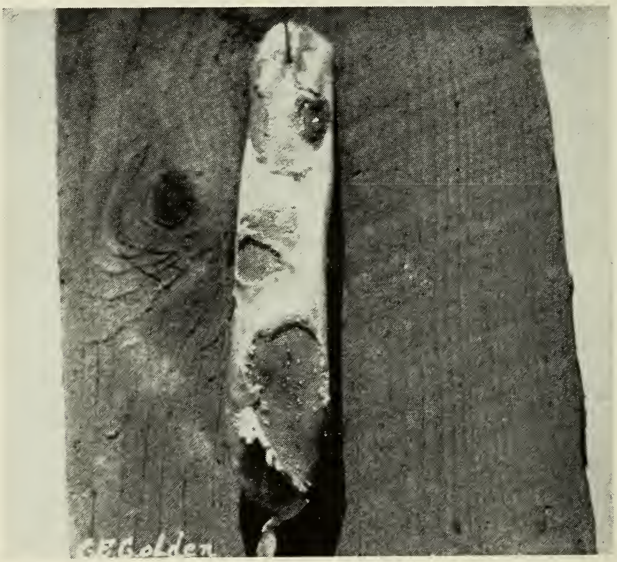
The virus of foot-and-mouth disease is of historic interest. Loeffler and Frosch (5) in 1897 showed that it would pass through clay filters so fine that ordinary bacteria were held back. Thus was demonstrated for the first time the so-called "filterable viruses" as a source of contagion for man and animals. The virus may remain alive and active for 6 months if kept moist and cool at a temperature between 3.5° and 5.5° C. and for 105 days when dried rapidly in a vacuum at room temperature. Heating at 55° C. for 15 minutes destroys it. It withstands bichloride of mercury 0.1 per cent or cresol 3 per cent for six hours. It is quite resistant to certain chemical disinfectants, as ether, alcohol or chloroform, but Olitsky has shown that this was because of a protective precipitation of protein around the molecule. When an alkaline solution is used, such as sodium hydrate in 1 or 2 per cent, to prevent coagulation of the protein, the virus is quickly destroyed.

Nearly a score of organisms have been described as the causative agent of foot-and-mouth disease, none of which have proved to be the case. Recently Frosch and Dahmen, working in Germany, claimed to have cultivated the organism, which they named *Loeffleria nevermanii,* but their results could not be confirmed by either the American or British Commission. The virus is of such minute size that it cannot be thrown down with a centrifuge. Material diluted 10,000,000 times is still capable of infecting guinea pigs.

The American Commission to study Foot-and-Mouth Disease have drawn attention to the close similarity of the virus of foot-and-mouth disease to that causing vesicular stomatitis in horses (4). In each instance the virus may be propagated in guinea pigs in continuous series; the experimental disease produced is very similar and may be transmitted to cattle and swine. The selective filterability of the two viruses show parallel responses. Immunologically, however, clear-cut distinctions were noted. There was no cross-immunity in guinea pigs, cattle or hogs, while horses failed to respond to inoculation with the virus of foot-and-mouth disease, although highly susceptible to the virus of vesicular stomatitis.

The Disease in Animals

Foot-and-mouth disease is the most contagious infection known to veterinary science. Cattle are very easily infected by a single exposure. One incident is related in which an infected lost calf trying to find its mother, spread the disease to twenty small dairy herds. The incubation period may range from eighteen hours to three weeks, usually being from three to six days. The disease first indicates itself by fever. Two or three days later small blisters appear on the mucous membranes of the mouth. Then the feet become swollen and tender and vesicles appear as in the mouth. The animal becomes lame, refuses to eat because of pain, and if a milch cow, almost dries up its flow of milk. The death rate is small in this form of attack and the animal will regain its normal appetite in 10 to 20 days. The full flow of milk is retarded for a long time however. More malignant types of the disease attack the internal organs and quickly cause death or leave the animal permanently injured. Among young animals the death rate may be 60 to 80 per cent, as a result of gastroenteritis or endocarditis.



United States Department of Agriculture

Fig. 7.—Lesions on tongue in foot-and-mouth disease.

The vesicles which form in the course of the disease contain the virus in concentrated form. When those in the mouth rupture, the saliva becomes infected and thus infection is spread to manger and pasture, food and water. Virus from vesicles on the feet is trailed wherever the animal goes, while that on the udder may contaminate the hands of persons coming in contact with the animal and thus be spread to other animals.

Human carriers of the virus of foot-and-mouth disease are responsible for a certain per cent of the cases among cattle. Direct contact with infected animals is the most important factor, while indirect contact follows closely. In the 1924 and 1925 outbreaks in California and Texas, Mohler gives the probable mode of infection of 551 herds (table 20).

The Disease in Man

Man may become infected with foot-and-mouth disease by eating or drinking raw milk products from infected cows. Children especially may become infected through drinking unpasteurized milk. The salivary secretions of animals may cause infection in man by entering directly into the mouth, as from a finger put into the mouth. Persons in charge of diseased animals not uncommonly become infected in this manner. While it is possible to become infected by cutaneous inoculation this is not the usual manner. In the 1908 epidemic when infected vaccine virus was distributed, there is no record of a human case occurring from the source. Gins (6), on the contrary, records the case of Pape who contracted the disease through a wound on the hand caused by the breaking of a flask filled with vesicular contents.

The symptoms in man are fever, vomiting, painful swallowing, sensation of heat and dryness of the mouth followed by an eruption of vesicles distributed over the mouth, lips and tongue. The hands may also be affected. Headache, pain in the back, vertigo, colic, diarrhea and weakness accompany some cases. Very seldom is the disease fatal, except in frail children, when an intestinal catarrh may cause death. Usually, however, the symptoms are very mild, even too slight to require the attendance of a physician. Siegel reports an epidemic with 8 per cent mortality.

Not many cases of the disease in man are on record in this country. In the 1870 epidemic Law observed human infection from drinking infected milk. In the 1880 and 1884 outbreaks no instance of transmission to man is recorded. In 1902, 1908 and 1914 a few cases of milk-borne origin were reported.

The American Foot-and-Mouth Disease Commission (7) emphasizes the fact that man is rather resistant and rarely contracts the disease. None of the human cases cited in the literature, with few exceptions, have been verified by inoculation of susceptible animals. In many of such reported cases therefore, the validity of the diagnosis can be reasonably questioned.

Preventive Measures

Preventive measures against the spread of the disease in cattle consist in the immediate destruction of all animals exposed to infection, in the elimination or destruction as far as possible of all wandering animals that might act as carriers, in rigid quarantine against visitors from infected districts, and in the careful disinfection of premises where disease has existed. Persons leaving such premises should be carefully disinfected as to clothing.

Pasteurization of milk is a simple and effective safeguard for the public against infection. Milk, butter and cheese from infected or suspected areas should receive like treatment. For those persons who must come into contact with infected animals, as veterinarians, farmers, etc., the ordinary precautions of personal hygiène will go far towards protecting them.

Items of Note

The unique features of foot and mouth disease are several.

1. It is the most highly contagious disease to which cattle may be exposed.
2. It is not particularly infectious for man, children being more susceptible than adults.
3. The death rate in man is very low.
4. The virus has certain similarities with that of vesicular stomatitis of horses.
5. The eradication of foot-and-mouth disease has been accomplished in the United States only by the most drastic and complete destruction of infected and exposed herds.
6. It can be said that in the United States foot-and-mouth disease does not exist, either as an agricultural or public health problem.

Table 20

HERDS SLAUGHTERED IN CALIFORNIA AND TEXAS IN WHICH PROBABLE MODE OF INFECTION WAS REPORTED (1)

Mode of Infection	California 1924	Texas		Total
		1924	1925	
Animals brought direct from infected public stockyards for slaughter, feeding, breeding or dairy purposes	4			4
Animals brought from infected stables or lots of local dealers	3			3
Direct contact with neighboring infected herds by pasture, mating, breeding animals or animals not confined	196	8	142	346
Infected railway cars	2			2
Persons visiting, exchanging work, stock buyers, peddlers, etc.	67	2	5	74
Dogs, poultry, birds, etc.	5			5
Infected public highways	16			16
Contaminated streams or drinking water	2			2
Infected feed or garbage	6			6
Other known sources of infection	7			7
Sources of infection unknown	79	1	6	86
	387	11	153	551

BIBLIOGRAPHY

1. Mohler, J. R.
U. S. Dept. Agri. Dept., Circ. 400, Dec., 1926.
2. Mohler, J. R.
U. S. Dept. Agri., Farmer's Bull. 666, Apr., 1915.
3. Olitsky, P. K., and Boëz, L.
Jour. Exp. Med., 1927, *45,* 673-683, 685-699, 815-831, 833-848.
4. Olitsky, P. K.
Jour. Exp. Med., 1927, *45,* 969-981.
5. Loeffler, and Frosch
Cent. f. Bakt. Abt. 1, 1898, *23,* 371.
6. Gins, H. A.
Klin. Wochenschr, 1924, *3,* 1135.
7. Olitsky, P. K., Traum, J., and Schoening, H. W.
Technical Bull. 76, U. S. Dept. Agri., 1928.

CHAPTER IV

MALTA FEVER AND CONTAGIOUS ABORTION

Malta fever is primarily an infection of goats caused by *Brucella melitensis,* characterized by no clinical signs which would lead to a definite diagnosis. Contagious abortion is primarily a disease of cattle and hogs caused by *Brucella abortus,* characterized by inflammatory changes of the mucous membrane of the uterus and fetal membranes resulting as a rule in the premature expulsion of the fetus. The goat, cow and hog organisms are probably all descendants from a common parent strain.

These diseases are secondarily infections of man, the symptoms in each case being characterized usually by a protracted febrile condition with a tendency toward recurrence. Indeed the symptoms are so inconstant and confusing that no one of them can be said to be typical, and a differential diagnosis is almost impossible in most cases without serological aid.

A multitude of names have been used to describe these conditions in man. The following terms for Malta fever are found in the literature associated with the geographical distribution:

Malta fever, Maltese fever, Mediterranean fever, Danubian fever, Neapolitan fever, Rock fever, Gibraltar fever, Cyprus fever.

From the clinical manifestations, the terms undulant fever and febris undulans have been used because of the wave-like accessions of fever, and Mediterranian phthisis from the bronchitis, anemia and night sweats suggestive of tuberculosis. Other similar names suggesting clinical manifestations are Bruce's septicemia, melitensis septicemia, febris melitensis, and bilious remittent fever. In Texas and New Mexico the terminology has differed somewhat—slow fever, goat fever, continued fever, mountain fever, Rio Grande fever and dust fever (because it was supposed infection was contracted in the dust filled goat pens.) The diagnosis is often confused with other diseases which has led to such designations as intermittent typhoid fever or typho-malarial fever. The diseases for which undulant fever is mistaken are typhoid, paratyphoid, malaria, tuberculosis, rheumatic fever, influenza, septicemia and sometimes kala-azar and amoebic liver infections.

The relationship of contagious abortion of cattle and of hogs to human infections has but recently been recognized. Such infections are much more rare than are the goat infections, hence no nomenclature has arisen to designate them. The diagnosis has usually been mistaken for other diseases, as tuberculosis, typhoid fever, malaria, influenza, rheumatic fever or articular rheumatism, until laboratory examinations of blood serum have proved otherwise.

The term "undulant fever" is found in the recent literature to designate human cases infected with any of the three organisms and is generally accepted all over the world to describe this form of infection. The government of Malta objects strongly to the term "Malta Fever."

History

Undulant fever has probably existed for many centuries. Hippocrates described a disease in man characterized by protracted fever with a tendency to relapse, which somewhat resembled tuberculosis and which was not very fatal. It is probable that the disease was Malta fever. Nothing further is recorded of it, however, until 1814 when Burnett described it under the name of "remittent malarial fever."

In 1863 Marston described it in detail, calling it "Mediterranean remittent fever." In 1886 Bruce isolated the causative organism, which he named several years later (1893) *Micrococcus melitensis.* In 1897 Wright and Semple described the agglutination test for Malta fever, resulting in a distinct advance in the diagnosis of the disease. The first accurate knowledge of its epidemiology, connecting the disease with goats was the result of the study of the British Commission for the Investigation of Mediterranean Fever. Six reports were put out during the years 1905-1907. It was shown that in the Island of Malta goats milk was the usual source of infection in man; 10 per cent of the goats were found to be excreting the organisms in the milk. Further tests showed 50 per cent of the animals were infected, and infection of the milk was very common through dirt from the abnormally long udders that often touched the ground.

Contagious abortion likewise probably has a record of great antiquity. The disease in Europe has been known for centuries where the infectiousness of the malady in cattle, together with the losses caused by it are related by Moscall in 1567, by Lawrence in 1805 and by Skellet in 1808. The "Complete Farmer" in 1807 stated that "it is considered certainly contagious, and when it happens the abortion should be immediately buried, and the cow kept as widely apart as possible from the herd, and not receive the bull that goes with them." Numerous other investigators added to the knowledge of the disease during the next century—Jonati in 1837 and Barlow in 1851. St. Cyr in 1875 stated his belief in a specific agent and the next year Franck communicated the disease to healthy cows by introducing the discharge and fetal membranes from aborting animals. This work was corroborated by Lehnert (1878) and Bräuer (1880). Nocard in 1888 isolated two organisms—a short bacillus and a micrococcus—but failed to reproduce the disease in healthy animals with either one.

Bang, assisted by Stribolt, in 1896 discovered the etilogical agent, which was named *Bacillus abortus.* The disease could be reproduced at will with pure cultures of this organism when administered to susceptible animals, and from the uterine discharges the same organism recovered. Numerous investigators since that time have confirmed these findings.

The agglutination test as well as the complement fixation test was first used by several investigators for the diagnosis of the disease in cattle in 1909 and 1910, following the description of similar tests for the diagnosis of glanders in horses. Among these were McFadyean and Stockman, Holth, Grinstead, Wall, Zwick and Brüll.

The date that contagious abortion was introduced into the United States is not known, but it must have been quite early in the history of the colonies along with imported cattle.

The first suspicion that it was related to human health was the demonstration of abortus organisms in cows milk by Schroeder and Cotten of the United States Department of Agriculture in 1911. The same year Mohler and Traum, co-workers of the above investigators, published the results of the first isolation of the organism from the tonsil of a child. In 1913 Larson and Sedgwick showed the presence of agglutinins and complement fixing bodies in the blood stream of children. In 1918 Dr. Alice Evans at the United States Department of Agriculture demonstrated the close identity of *Bacillus abortus* of cattle and *Micrococcus melitensis* of goats and later the relationship of the abortus organism to human infection.

THE INFECTING ORGANISMS

One of the strange occurrences in the history of bacteriology was the failure over a period of more than twenty years to identify two organisms which are so closely related as to be almost indistinguishable. Indeed not only was the relationship overlooked but two different generic names were used to designate them, this in spite of the fact that the resources of bacteriologists were later taxed to the utmost to find distinguishing characteristics.

Micrococcus melitensis was isolated by Bruce, working in the Island of Malta in 1887, as the causal organism of Malta fever of man. Ten years later Bang in Denmark discovered *Bacillus abortus* as the cause of infectious abortion of cattle. The differences in geographic distribution of the two diseases, in the animals primarily affected, in the clinical manifestation of the diseases and finally in the fact that Bruce incorrectly described his organism as a micrococcus, all served as definite barriers against a comparative study of the two organisms.

Alice Evans in 1917, working in the Dairy Division of the United States Department of Agriculture, began the study which has since proved so valuable in both veterinary and human medicine. She was making a study of the bacterial flora of cow's milk when the problem of *Bacillus abortus* was confronted. During a conversation with Dr. Eichhorn, Chief of the Pathological Division, it was suggested that a comparative study of *Bacillus abortus* and *Micrococcus melitensis* be made. This was done with the astonishing results that the two organisms were indistinguishable except by agglutinin absorption tests.

The nomenclature of the two organisms is confusing. Following are some of the designations:

Micrococcus melitensis by Bruce 1887	*Bacillus abortus* by Bang 1897
Bacillus melitensis by Jordan 1912	*Corynebacterium abortus endemici* by Preisg 1903
Bacterium melitensis by classification of Society of American Bacteriologists 1918	*Bacterium abortus* by classification of Society of American Bacteriologists 1918
Alcaligines melitensis by Castellani and Chalmers 1919	*Alcaligines abortus* by Castellani and Chalmers 1919
Brucella melitensis by Meyer and Shaw 1920	*Brucella abortus* by Meyer and Shaw 1920

The term *"alcaligenes"* has been accepted by the Society of American Bacteriologists and is used in Bergey's Manual of Determinative Bacteriology. On the other hand the term *"Brucella"* has gained popular approval both in Europe and America, being used by Evans, Carpenter, Huddleson and many others in the periodical literature. The removal of this group of pathogens from the other non-pathogenic members of the *Alcaligenus* genus is strongly recommended by Evans. The new genus, according to the demands of priority would be termed *"Brucella,"* designated by Br.

The outstanding features of the organisms are their great pleomorphism, sometimes assuming the form of cocci (which misled Bruce) and at other times the form of bacilli and the difficulty of separating strains of human, bovine, porcine, caprine or equine origin by morphological, cultural, biochemical, pathological or serological means.

In primary cultures the *melitensis organism* of caprine origin is rather easily grown by the ordinary laboratory methods, while the *abortus organism* of bovine origin requires an atmosphere of 10 per cent carbon dioxide for the best results. After twelve to fifteen transfers on culture media it will grow at atmospheric CO_2 tension.

In addition to the bovine and caprine strains of this group of organisms there exists a porcine strain which is closely related but which has a special virulence for swine.

The term *parmelitensis* was suggested by Nègre and Raynaud to designate those strains which were similar to *melitensis* in morphological, cultural and biochemical behavior but failed to agglutinate, or agglutinated only slightly, in *melitensis* serum. *Paramelitensis* organisms absorbed agglutinins from *Melitensis* serum only partially.

A serological classification of members of the *melitensis-abortus* group was made by Evans. Among 68 strains of organisms from human, bovine, caprine, porcine and equine sources, eight different groups were found by means of agglutinins absorption test. The report concludes (1)

> "The majority of bovine and porcine strains fell into one large group (33 strains) which is designated variety *abortus*. Five strains of human origin were of this variety.

"Another group important in this country includes strains of human, bovine, caprine and equine origin (12 strains). It is designated variety *melitensis* A.

"Three groups which were found to be prevalent in the Mediterranean countries did not occur among the strains received from countries outside of those regions. One of those groups is designated variety *melitensis* B; another, which corresponds with the descriptions of the so-called *paramelitensis* is designated variety *paramelitensis;* another serologic group is designated *para-abortus* because it is serologically closely related to the *abortus* variety, and exhibits agglutination peculiarities like those of the variety *paramelitensis.*"

Simple agglutination tests cannot distinguish these different varieties and groups.

It is evident from the close relationship of these various groups that the parent strain was the same for all. Long years of parasitism in different hosts has rendered but slight change in any of the characteristics except virulence for certain animals.

Geographical Prevalence

Malta fever occurs among goats and man in many of the subtropical countries—in countries bordering the Mediterranean, in South Africa, China, the Philippines, Peru and in Arizona, Texas, and New Mexico in the United States. It has been said that the disease does not exist in Europe north of 46° lattitude. This statement is not exactly correct, as outbreaks have been known to occur farther north. Evans draws attention to the curious fact, however, that, although goat's milk is widely used in Hungary, Switzerland, Czechoslovakia and Southern Germany, the prevalence of Malta fever cannot be compared to that in the neighboring countries to the south.

Contagious abortion among cattle is widespread, but more particularly is it an affliction of temperate climates—the British Isles, France, Switzerland, Germany, Denmark, Belgium, Austria, South Africa, the United States and Canada. Possibly the cause of this is because the cattle industry is more particularly confined to these areas. The warmer countries are also afflicted, however, as reports have come from Italy, Palestine and the Dutch East Indies. Infections in man from bovine sources, of course, follow the distribution of the disease in cattle.

Outbreaks of abortion in swine, according to Theobold Smith (2), are confined to the middle west and the western coast of the United States.

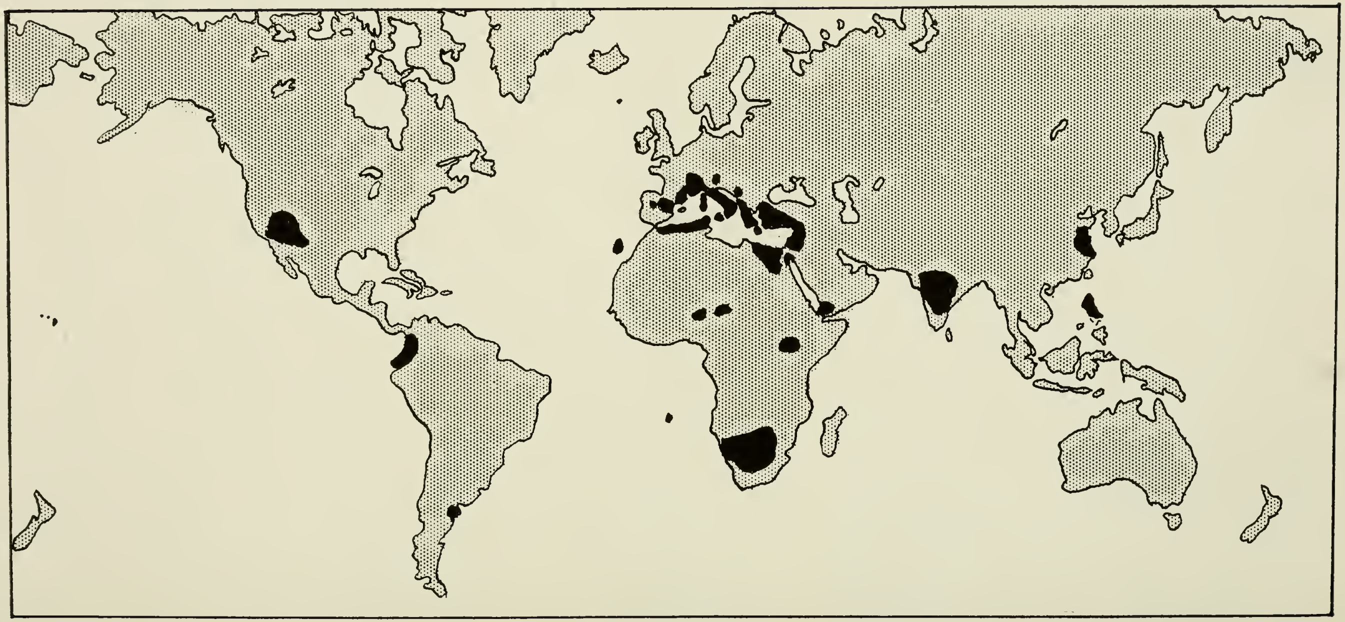


Fig. 8.—World distribution of undulant fever in man of goat origin (after Castellani). In the United States there have been many cases along the Mexican border, but very few in the eastern part of the country.

Table 21

SUSCEPTIBILITY OF ANIMALS TO Br. MELITENSIS AND Br. ABORTUS
(Huddleson)

Species	Melitensis		Abortus (bovine)		Abortus (porcine)	
	Large D	Small D	Large D	Small D	Large D	Small D
Man	++++	++	++++	−	++++	−
Monkey	++++	++++	++++	?	++++	++++
Bovine	++++	++++	++++	++	++++	?
Goat	++++	++++	++++	++	?	?
Swine	?	?	++++	−	++++	++
Rabbit	++++	++++	++++	++++	++++	++++
Guinea pig	++++	+	++++	++	++++	++++
Rats	?	?	++++	+++	?	?
Mice	?	?	++++	+++	?	?

Large D = large dose; Small D = small dose.

SEASONAL PREVALENCE

There is probably no seasonal prevalence for this group of diseases, either in cattle or man. The cases of infection of man from bovine sources which I have encountered have come in during all months of the year.

Malta fever of caprine origin is said to be more prevalent in man during the spring and early summer months, not only because of the greater use of goats milk during that period, but also because of the fact the milk is contaminated to a greater degree following the birth of the kid in the spring.

MALTA FEVER IN ANIMALS

Malta fever is primarily an affection of goats. The animals show no morbid symptoms of the disease, however, and unless man had been infected the disease in goats would never have been suspected. Animals, fat and in apparently good health, will show positive agglutination reactions in the blood during life and at post-mortem there can be demonstrated the *melitensis* organisms in the tissues. In some instances, when goats die of the disease, there is congestion and degeneration of the liver, hypertrophy of the spleen and swelling of the abdominal lymphatic glands and mastitis. At other times infected animals exhibit indefinite symptoms such as lassitude, loss of flesh, cough and lameness. Dubois states that abortion may take place in 50 to 90 per cent of the animals when a flock is first infected. *Brucella melitensis* is present in the milk, urine, blood and tissues of infected goats. The excretion in the milk may be intermittent, disappearing for a week or more at a time, then reappearing in enormous numbers.

Sheep rank close to goats in susceptibility. Césari (26) recounts that 49 out of 429 sheep at Aveyron gave positive agglutination tests, while in two other instances 7 out of 74 and 6 out of 12 were positive.

Horses are rather resistant to infection with *melitensis.* In Sicily 20 per cent of the horses gave feeble agglutination reaction, while Dubois reports 2 per cent positive tests in horses. This does not indicate, however, actual cases of infection. A large percentage of horses will agglutinate the organism slightly.

Mules are much more susceptible than horses. In the Island of Malta, Kennedy found 37 positive agglutination reactions among 87 mules. Césari tells of two men taking care of such infected mules, both of whom contracted Malta fever; neither of the men had come in contact with goats.

Cows have been found naturally infected in the Island of Malta, excreting organisms of *melitensis* in the milk. Evans inoculated a pregnant heifer with a culture of the *melitensis* organisms, inducing abortion and obtaining the organism from the fetus and colostrum.

Dogs may give an agglutination reaction to *melitensis* normally as high as 1 :600. In the Island of Malta, Kennedy found 9 such reactions among 114 dogs. From one he isolated the *melitensis* organism from a mesenteric gland. Bruce found among 22 cats, 5 positive reactors, from the mesenteric gland of one of which he isolated *melitensis.*

Rabbits, guinea pigs, rats and mice, are susceptible to artificial infection but are not often found naturally infected. The infection of poultry is doubtful according to Césari. He cites, however, several instances of positive agglutination reactions in hens, chickens and ducks, and one instance where *Br. melitensis* was isolated from the spleen of a hen.

Torstenson (27) observed that cats, dogs, guinea pigs and gray rats kept in contact with infected goats gave negative evidence of infection, indicating that such animals rarely contract the disease naturally, although subject to artificial infection.

Monkeys as well as man easily acquire the disease.

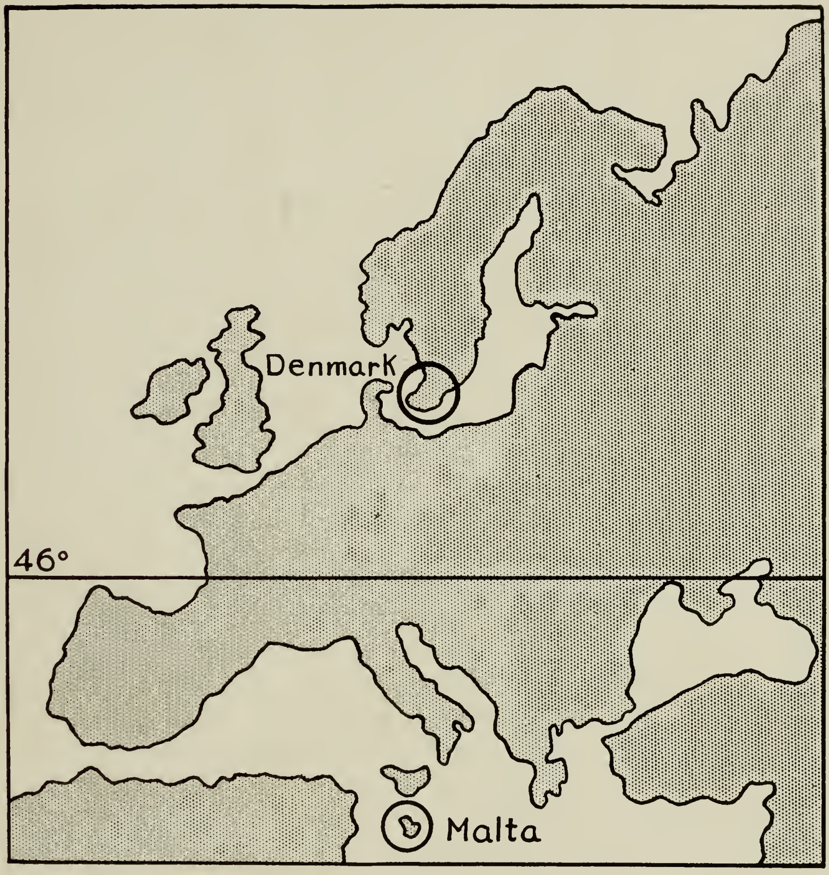


FIG. 9.—Although the organisms of Malta fever of goats (discovered in Malta) and contagious abortion of cattle (discovered in Denmark) are so closely allied as to tax the efforts of bacteriologists to separate them, the difference in geographical distribution as well as the animals involved, prevented suspicion of the relationship. Undulant fever of goat origin does not prevail to a great extent north of 46° latitude.

Malta fever, as an animal disease problem in the United States, is confined to goats in Texas, Arizona, and New Mexico, where it has existed probably for several decades.

The first experience with the disease in this country was in 1905. The United States Department of Agriculture delegated one of its members to go to Europe to procure a supply of goats with which to build up the milk goat industry. After inspecting several sources, Dr. Thompson selected sixty-one females and four males from the Island of Malta, arriving with them in New York, in September, 1905. During this time the British Commission for the Investigation of Mediterranean fever had established the fact that Maltese goats were carriers of the infection of Malta fever. As soon as the British Commission learned of the importation of such goats into the United States, it informed the Secretary of Agriculture of the danger. The goats were immediately quarantined upon their arrival. An investigation made by Mohler and Hart upon the urine and milk as well as agglutination tests of the blood revealed the fact that several animals were infected. Efforts to eliminate the disease from the herd, so that some of the goats might be saved, were unsuccessful and eventually all were destroyed.

The fact that Malta fever existed endemically in the United States was not established until 1911 when Gentry and Ferenbaugh (3) discovered the disease in humans in the goat-raising section of Texas, tracing the infection to goats, the blood of which gave positive agglutination tests. Mohler and Eichhorn (4), who confirmed the above observations, estimated that Malta fever had existed in Texas and New Mexico for at least twenty-five years. In 1925 Holt and Reynolds (5) of the United States Army investigated twenty-two counties in Texas, New Mexico, and Arizona along the international boundary. In seven of these counties no goats were found, but in the other fifteen Malta fever was demonstrated. Four counties north of the border counties were likewise found to harbor infected goats.

Contagious Abortion in Animals

Contagious abortion in cattle is the most serious disease, next to tuberculosis, with which the animal industry has to deal. In the United States it exists everywhere, in range cattle as well as dairy cattle. The economic loss resulting from its ravages is estimated by Edwards and Coffee (30) to be eighty millions of dollars annually.

The date that contagious abortion was introduced into the United States is not known, but it must have been quite early in the history of the colonies along with the importation of infected cattle from Europe. It apparently spread rather slowly at first, but of late years has found its way to almost every farm and range.

In Missouri, over a period of ten years, 15,000 cattle were tested for contagious abortion, 30.22 per cent of which gave positive agglutination reactions (28). At the Storrs Experiment Station in Connecticut 29.1 per cent of cattle purchased reacted to the disease. Ninety per cent of the herds in that state are said to be infected. In Pennsylvania, according to Munce, 65 per cent of the cows that are sent to the butcher on account of their failure to reproduce are sent because of contagious abortion. In Michigan 30 per cent of the cattle are reactors to the agglutination test, but, as Huddleson points out, all such animals may not be infected. In Illinois about 10 per cent of cattle reacted in 55 herds tested by Graham. The Committee on Bovine Infectious Abortion of the American Veterinary Medical Association lists six states as practically free from infection—North Dakota, Wyoming, New Mexico, Alabama, Mississippi, and West Virginia (6).

The disease in cattle has no particular clinical symptoms except those associated with the act of aborting. The health of the cow does not seem to be impaired and the death rate is nil.

The most serious consequences of infection are diminished milk supply, either through a lessened flow due to the diseased condition of the udder or being dry for a long period; loss of calves; sterility, either temporary or permanent; and septic conditions of the uterus or udder disorders due to parturition occurring while the glands are actively secreting. *Brucella abortus* has an affinity for embryonic tissues and is present in the uterus of infected pregnant animals. After abortion it soon disappears from the uterus but may still be found in the udder. Here it may remain several years causing serious damage to the milk producing tissue and appearing regularly in the milk. It is also found in the uterine discharges accompanying abortion.

Bulls are susceptible to infection at times. Buck, Creech, and Ladson (7) found the seminal vesicles of 4 bulls infected out

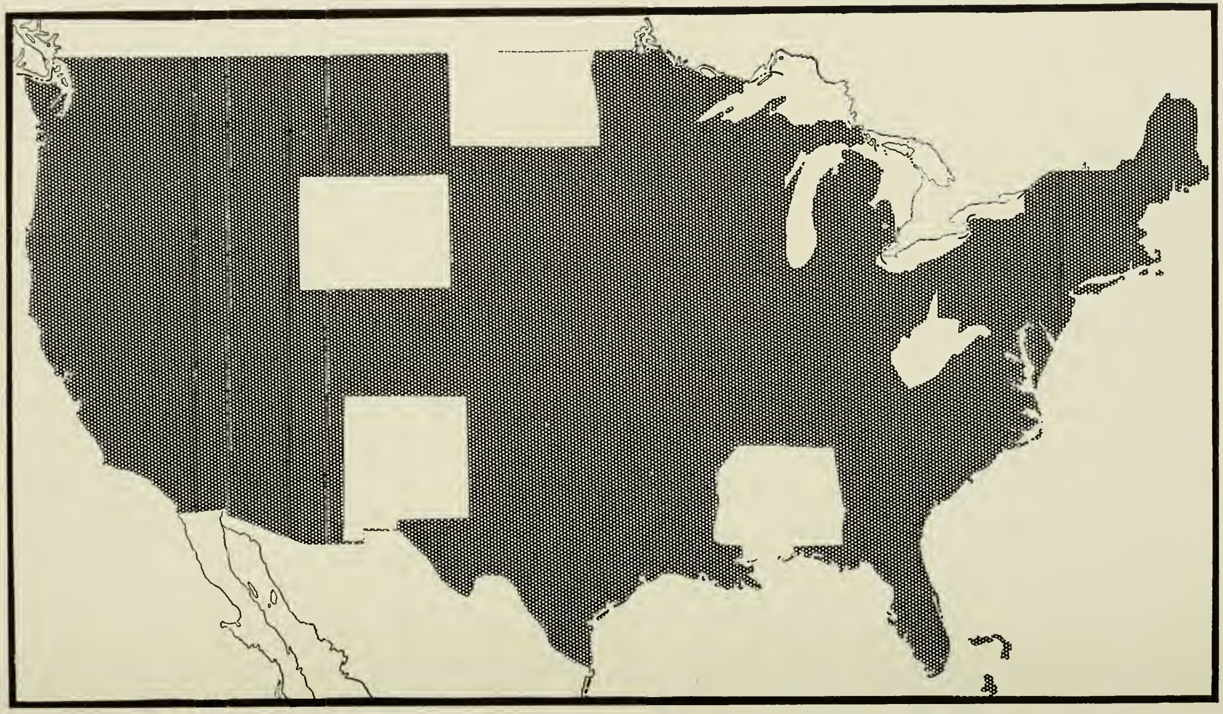


Fig. 10.—Contagious abortion in cattle in the United States prevails extensively. Six states in 1926 were found relatively free.

of 325 examined. The lesions are often so extensive that permanent sterility results.

Infection of swine with contagious abortion was first noted in 1914 by Traum and confirmed in 1916 by Good and Smith (8), from which time it has become increasing prevalent in the United States, especially in the Middle West and on the west coast. The strain of *abortus bacillus* responsible for porcine infections is more highly virulent for hogs than is the bovine strain.

Sheep and goats have been reported by Schroeder as susceptible to infection with contagious abortion through contact with cattle. The animals abort as do cattle. Foxes likewise have been reported by MacFadyean and Stockman as aborting when they have come into contact with the infection.

Horses are subject to an infection causing abortion which should not be confused with the type of infection discussed here. *Salmonella abortus-equi* is a member of a distinctly different group of organisms. One case is on record of infection with the bovine organism (1) and one with the porcine organism.

The control and prevention of contagious abortion in cattle is of vast importance economically. The extent of infection in any herd may be determined by means of the agglutination test on the blood serum of each animal. A single test, however, is not sufficient to discover all animals harboring the germs. Several weeks or months may elapse after an animal has been infected before it shows a positive agglutination reaction. The test must be repeated two or three times, therefore, at intervals of six weeks or two months, before all infected animals are eliminated.

Herds that are badly infected, showing more than half the animals as positive reactors, cannot well be cleared of the disease. The most economical method for the owner is to dispose of the entire herd and start new with uninfected stock.

Herds that show only a few reactors can be cleared of infection by eliminating the affected animals and carefully and thoroughly disinfecting the premises. Subsequent tests should be made to pick up any animals that may have been missed on the first test.

Herds that are entirely free from infection may be kept in that condition by strict attention to certain details. The disease is usually introduced through the purchase of infected cows or pregnant heifers. Bulls and unbred heifers are said to be of less danger in spreading the infection. The rôle of the infected bull at the time of service has been much discussed. It is maintained by observers that if the service takes place on neutral ground there is small chance of the cow becoming infected. The infected semen from the bull or vaginal discharge from the cow contaminating the premises is of greatest significance. The agglutination test should be used before any new animal is admitted to an uninfected herd. Other routes of infection are hay and grain produced on farms where the disease exists, drainage from an infected pasture, bedding from railroad stock cars scattered along the track and neighboring pastures and unpasteurizezd milk from creameries used to feed calves.

The use of vaccines to immunize cows against contagious abortion has received much attention, but results so far are disappointing. Numerous methods of preparation and administration have been tried but the immunity obtained has not been sufficient to afford complete protection against the disease.

Human Infections

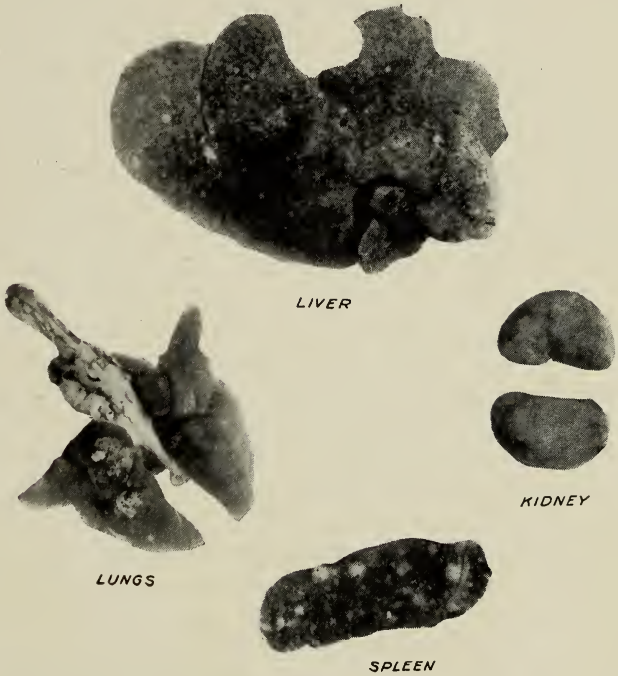
Malta fever in man, according to Bruce, is a "disease of long duration, characterized clinically by continued fever, profuse perspiration, constipation, frequent relapses, rheumatic or neuralgic pains, swelling of joints or orchitis; bacteriologically by the presence in the blood and organs of *Micrococcus melitensis;* anatomically by congestion of the spleen and other organs." These symptoms, however, are so inconstant that, as Craig states (9), "no one of them can be said to be typical of the disease. A differential diagnosis is almost impossible in the majority of cases without the aid of the microscope and the sèrum test."

The human disease caused by *Brucella melitensis* in no way differs from that caused by *Brucella abortus,* so far as known, except the latter is much more rare. The symptoms of one have no special characteristics to distinguish them from symptoms produced by the other type.

The *abortus bacillus* has not been found ordinarily to locate in the genitals of the human race. Belyea (10), however, describes one such case in which the *abortus bacillus* of bovine type was isolated from the vaginal discharge. No cases of abortion in women are recorded.

The incubation period is usually about two weeks but may be shorter or longer. The patient becomes aware of general malaise, muscular pains and fever which is usually higher in the afternoon. The course of the disease may drag along for some time, being mistaken for any one of several infections such as tuberculosis, malaria, or typhoid fever. Very often there are recurrences of fever after a lapse of several days or weeks of normal temperature. The seriousness of the trouble is readily apparent when it is realized that a permanent cure is unusual and difficult to obtain.

Undulant fever has been of considerable importance in past years as a human affliction in southern Europe and other semitropical climates. While the disease had been prevalent for centuries it was not until 1905 that the goat was definitely linked up with the affliction. Following out the suggestion of the British Mediterranean Fever Commission, Malta fever among the British military and naval forces stationed on the Island of Malta was entirely eliminated through the simple expedient of prohibiting the use of raw goat's milk. The prevalence of the disease in the



United States Department of Agriculture

FIG. 11.—Lesions in Guinea pigs caused by Brucella abortus.

civil population, on the other hand, continued unabated. Horrocks (11) states that Malta fever practically disappeared from Gibraltar since the practice of importing goat's milk was stopped. Stitt relates that on the ship which carried the original consignment of 65 goats from Malta to Antwerp, destined for the United States in 1905, several cases of Malta fever appeared among the crew due to milk from these infected animals. Two German engineers, however, who boiled the goat's milk, escaped infection.

Undulant fever of caprine origin in the United States has never been a serious public health problem. From time to time cases of infection originating elsewhere have appeared. Tyson describes a case in 1898, the source of infection being Cuba. In 1903 Craig described the first case which is known to have arisen in the United States. The patient was an Army nurse and it was presumed that the infection was contracted from caring for sick soldiers recently returned from the Philippines. It is entirely possible, however, that this was a case of bovine rather than caprine origin.

The first actual knowledge of endemic undulant fever due to goats in the United States resulted from the investigation of Gentry and Ferenbaugh in 1911 (3). It was established that the disease not only existed at that time in the goat raising territory of Texas, but also had existed for some time under such names as goat fever, slow fever, continued fever, Rio Grande fever, etc. Mexican goat herders attributed the fever to drinking raw goat's milk and escaped the disease by boiling the milk. Since that time sporadic cases of the disease have been reported from the Mexican border.

The first and only epidemic of undulant fever on record in this country of caprine origin occurred in 1922 in Phoenix, Arizona (13). In April of that year a herd of goats was brought to the city for the specific purpose of selling goat's milk. The industry was apparently quite popular, the demand rising to 830 quarts daily by June. During May, June, and July cases of an unknown febrile condition kept occurring until August, when the definite diagnosis of undulant fever was established and the sale of goat's milk prohibited. Among 400 or 500 people who had used goat's milk over a period of four months there were 40 known cases.

The relationship of contagious abortion of cattle to public health was a doubtful question for several years. Credit largely belongs to members of the United States Department of Agriculture for finally solving the problem.

In 1911 Schroeder and Cotton of the Bureau of Animal Industry were attempting to demonstrate tubercle bacilli in milk by guinea pig inoculation (14). Some of the injected animals showed lesions that might be mistaken for tuberculosis, but which contained no acid-fast bacilli. Other investigators had also encountered similar lesions. The peculiar malady was transmissable through a series of guinea pigs but its cause was not at first apparent. At last, however, there was isolated, after many attempts, *Bacillus abortus* of Bang. The organisms were shown to be quite common in milk and to be excreted over periods of several years' duration, by otherwise apparently healthy cows. Among seventy-seven samples of market milk, eight, or 10.5 per cent, harbored the organisms.

Mohler and Traum (15) in 1911 attempted to demonstrate the relation of contagious abortion to human infection by means of serological examination, but blood specimens from forty-two persons were negative. Of fifty-six adenoids and tonsils from children inoculated into guinea pigs, one specimen produced lesions in the experimental animal from which *Bacillus abortus* was isolated. Thus the first human infection was demonstrated.

Larson and Sedgwick (16) in 1913 made complement fixation tests on the blood serum of 425 children, finding 73 positive with *Bacillus abortus* antigen. Agglutination tests run on the same specimens gave parallel results. In a later report in 1915 they showed that in the original group of 425 children, those receiving milk from an abortion free herd were negative, while the children receiving mixed market milk gave a high percentage of positive reactions.

The demonstration of *abortus bacilli* in milk and immune bodies in the blood stream of children was confirmed by several investigators.

Cooledge (17) in 1916 found among fourteen persons, that of six who used raw milk three gave positive blood tests, of four using pasteurized milk one gave a positive blood test, while of four drinking no milk all were negative. It was discovered that agglutinins were present in the milk of one of the cows connected with the above study, and it was suggested that possibly the ingestion of antibodies from the cow by way of the milk were absorbed into the blood stream and were responsible for the positive tests. An experiment lasting over eight weeks, where seven persons consumed large quantities of milk rich in antibodies, failed to establish the contention.

Following the publication of Miss Evans' (18) report in 1918 on the identity of the *abortus* and *melitensis* organisms, interest in the possibility of human infection from contagious abortion of cattle was renewed, but it was not until 1924 that the first authentic case in man was described by Keefer (19). In rapid succession after that other human cases were reported till several dozen were accumulated in the scientific literature by Huddleson (20), Moore and Carpenter (21), Evans (1), Gage and Gregory (22), Knowlton (23), Hull and Black (24), and others.

The prevalence of human infection with the bovine strain of *abortus* is difficult to determine. It would appear that possibly it is more common than ordinarily realized. Among 69 blood specimens from fever patients which were submitted for the Widal test but found negative, Black and I obtained positive *abortus* agglutination tests and histories in five.

Several studies have been made to determine the exact status of the prevalence in the human population. Miss Evans obtained blood from 500 patients suffering from a variety of diseases, whose serum had been submitted for the Wassermann test. There were 59 giving positive reactions in the 1:5 dilution but not above 1:40. One patient gave undoubted evidence of infection, being positive in 1:320 dilution. The others were regarded as having ingested infected cow's milk which caused slight infections but not necessarily noticeable illnesses.

Litterer in Tennessee tested 1,000 blood specimens submitted for the Wassermann test, finding 107 to react in a dilution of 1:10 or greater. Carpenter in New York State tested 4,000 similar specimens of which 7 per cent were positive in high dilutions. McAlpine and Mickle in Connecticut, using routine blood samples submitted for the Wassermann test, studied 20,259 specimens, finding only 127, or 0.6 per cent, positive in 1:100 dilution. They conclude that "these results, obtained in a state where preliminary testing has shown at least 90 per cent of the dairy herds to be infected with *Bact. abortus* and approximately 60 per cent of the milk supply only is pasteurized, indicate that infection of man with the bovine type of *Bact. abortus* is relatively rare, provided any significance can be attached to the agglutination test" (25).

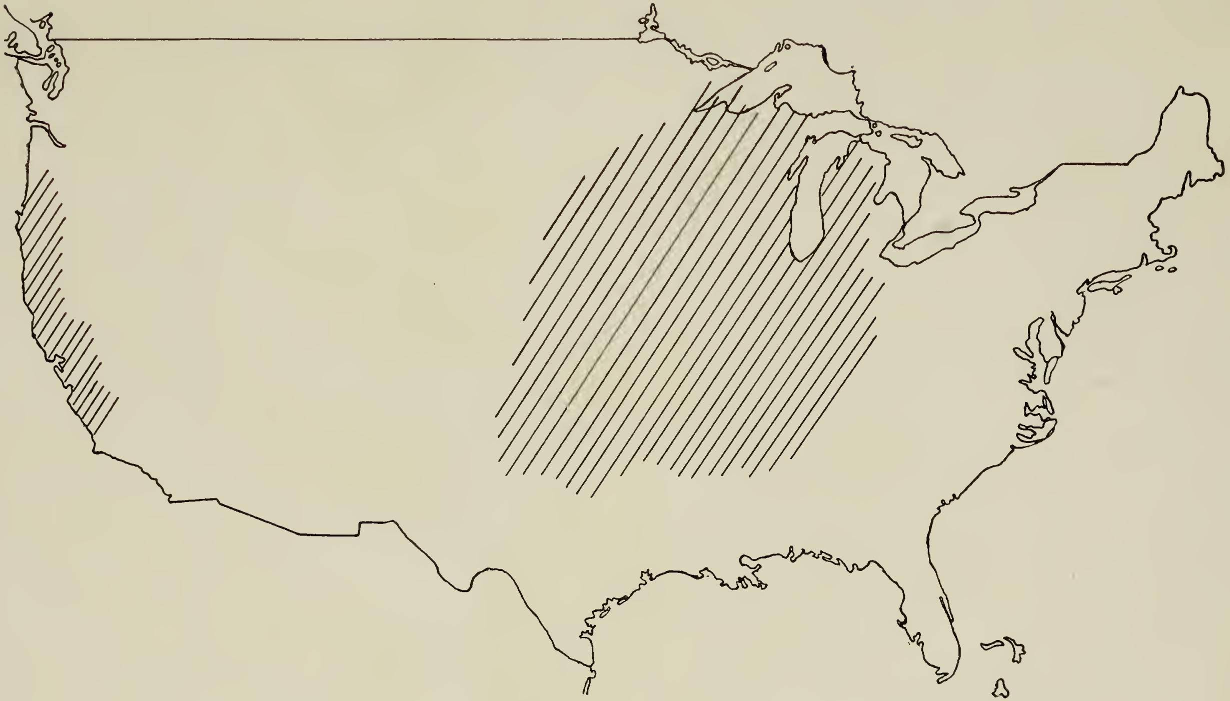


FIG. 12.—Contagious abortion in hogs is most prevalent in the United States in the Middle West and on the Western Coast.

Undulant fever is a disease of adults. Evans found only 4 cases in children under 9 years of age out of 55 studied. It is a disease of males, only 27 cases out of 117 being in females. It is a disease largely of rural sections, where milk is consumed unpasteurized or other contact with animals may occur, although many cases have been found in urban populations. Carpenter studied 155 cases, finding that 70 per cent had been drinking raw milk.

Hogs have been shown to be a source of human infection with the *abortus* organism. Miss Evans (1) reports two cases in 1924, one from South Dakota in a meat inspector who had been inspecting hogs for the last eight years, and one from Connecticut in a man who had been "shaving ears and quartering" hogs. McAlpine and Slanetz (29) have found all *abortus* cultures isolated from man in Connecticut to fall into the porcine group. They also call attention to the fact that the incidence of *Br. abortus* infection in man is higher in states where the swine industry is large. It is possible that cows may become infected with the porcine variety from close contact with swine, and thus pass on the infection.

Laboratory workers handling cultures of *abortus* or *melitensis* organisms have become infected from time to time. More than the ordinary precautions of laboratory hygiene are necessary when working with this group of organisms.

Inter-human transmission of undulant fever in Europe has been found negligible, with the disease never more than one remove from the goat. The same is probably true of the disease in this country of bovine or porcine origin.

Prevention

The protection of the public against infection with undulant fever, whether caprine, bovine, or porcine origin, is possible. All milk for human consumption (except certified) should be properly pasteurized. Man is not very susceptible to *Brucella abortus* of bovine origin—therefore the fact that the dairy herd is infected with contagious abortion is not sufficient cause to reject the milk from such animals for human consumption. A sufficient number of human infections occur, however, to require the destruction of the organism. This is easily accomplished by 30 minutes' exposure to a temperature of 142° F. Miss Evans states, "if there were no other reason for milk pasteurization, it would appear to be folly to drink raw milk containing the *abortus* organism."

The pasteurization of milk in country districts or on the farm where contagious abortion exists among the cattle is more difficult, for the process must be carried out on a small scale, usually by a busy housewife, in addition to a multitude of other duties. Boiling may be substituted for pasteurizing. The danger of infection is too great to be ignored.

The sale of goat's milk should be prohibited where Malta fever is known to exist. Man is very susceptible to infection with *Brucella melitensis.* Milk from goats shown by test to be free from the disease should be pasteurized as an added precaution. Milk from infected goats, when it is necessary to use it, should be boiled.

For those persons whose occupations expose them to infection, such as laboratory workers or employees at abattoirs where hog carcasses are handled, only eternal vigilance in personal hygiene will protect them.

Items of Note

1. The *abortus-melitensis* group of organisms is widely distributed over the world.
2. The common parentage of caprine, bovine, and porcine strains is indicated by the close identity of all characteristics.
3. The caprine strain, *Brucella melitensis,* residing long years in the goat as a host, has developed virulent properties for man.
4. The bovine strain, *Brucella abortus,* residing long years in the cow as a host, infects man at rather seldom intervals.
5. The porcine strain, *Brucella abortus,* is very pathogenic for man. It is more virulent than the bovine strain for all animals.
6. Malta fever of goats is a problem of semi-tropical countries. Its significance in the United States is relatively small, being confined chiefly to the Mexican border.
7. Contagious abortion of cattle is a problem chiefly of all climates. It is of tremendous economic importance in animal industry in the United States.
8. Protection of the public against undulant fever is easily afforded through proper pasteurization of the milk.

BIBLIOGRAPHY

1. Evans, Alice C.
   U. S. Public Health Service, Hygienic Lab. Bull. 143, 1925.
2. Smith, Theobald
   Jour. Exp. Med., 1926, *43,* 207-223.
3. Gentry, E. R., and Ferenbaugh, T. L.
   Jour. Am. Med. Asso., 1911, *57,* 730-731, 889-891, 1045-1048, 1127.
4. Mohler, J. R., and Eichhorn, A.
   28th Annual Report, U. S. Bur. Animal Ind., 1911, pp. 119-136.
5. Holt, R. L., and Reynolds, F. H. K.
   Mil. Surg., 1925, *56,* 414-424.
6. Report of Committee on Bovine Infectious Abortion
   Jour. Am. Vet. Med. Asso., 1927, 70, 875.
7. Buck, J. M., Creech, G. T., and Ladson, H. H.
   Jour. Agr. Res., 1919, *17,* 239.
8. Good, E. S., and Smith, W. V.
   Jour. Bact. 1916, *1,* 415-422.
9. Craig, C. F.
   Am. Jour. Med. Sci., 1903, *125,* 105.
10. Belyea, G. N.
    Jour. Am. Med. Asso., 1927, *88,* 1482.
11. Horrocks, H. W.
    Jour. Roy. Army Med. Corps., 1907, *8,* 374-384.
12. Tyson, James
    The Practice of Medicine. 2nd Edition, 1901.
13. Watkins, W. W., and Lake, G. C.
    Jour. Am. Med. Asso., 1927, *89,* 1581-1584.
14. Schroeder, E. C., and Cotton, W. E.
    U. S. Dept. Agri., Bur. of Animal Ind., Circ. 216, 1914.
15. Mohler, John R., and Traum, Jacob
    U. S. Dept. Agri., Bur. of Animal Ind., Circ. 216, 1914.
16. Larson, W. P., and Sedgwick, J. P.
    Am. Jour. Dis. Child., 1913, *6,* 326-333.
17. Cooledge, L. H.
    Jour. Med. Res., 1916, *34,* 459-467.
18. Evans, Alice E.
    Jour. Inf. Dis., 1918, *23,* 580-593.
19. Keefer, C. S.
    Bull. Johns Hopkins Hosp., 1924, *35,* 6-14.
20. Huddleson, I. F.
    Jour. Am. Med. Asso., 1926, *86,* 943-944.
21. Moore, V. A., and Carpenter, C. M.
    Cornell Vet., 1926, *16,* 147-152.
22. Gage, E. A., and Gregory, D. A.
    Jour. Am. Med. Asso., 1926, *87,* 848-849.

23. Knowlton, M.
Annual Report, Connecticut State Dept. of Health, 1924, 101-108.
24. Hull, T. G., and Black, L. A.
Jour. Am. Med. Asso., 1927, *88,* 463-464.
25. McAlpine, James G., and Mickle, F. L.
Am. Jour. Public Health, 1928, *18,* 609-613.
26. Césari, E.
Jour. Am. Vet. Med. Asso., 1925, *67,* 637.
27. Torstenson, H.
Arch. Inst. Past. Tunis, 1926, *15,* 305-308.
28. Connaway, J. W., Newman, H. G., and Uren, A.
Missouri Experiment Station, Bull. 244, 1926, pp. 58-61.
29. McAlpine, J. G., and Slanetz, C. A.
Sixteenth Annual Report of the International Asso. of Dairy and Milk Inspectors, 1927.
30. Jour. Am. Med. Asso., 1927, *88,* 407.

Chapter V

MILK SICKNESS

Milk sickness in man and trembles in animals results from poisoning with the white snake root or the rayless goldenrod. The disease is primarily one of animals transmitted to man through the meat, milk, or milk products of affected cattle. Other names by which milk sickness has been known are white snake root poisoning, slows, and puking fever in the East, and milk sick or alkali disease in the West.

History

Milk sickness was recognized as early as 1776 in North Carolina. It was a serious affliction of the early settlements of the Middle West, taking many lives in Tennessee, Ohio, Indiana, Illinois, Missouri, and other Mississippi River states.

Mather, in the "Making of Illinois," mentions it in connection with the settling of the "New Design" in 1797, stating that it attacked many newcomers. It was one of the diseases which gave Illinois the reputation of being unhealthful and so retarded immigration to this territory for awhile.

Herndon, in his life of Abraham Lincoln, describes the ravages of milk sickness in Kentucky, telling how whole settlements were abandoned to get rid of the peculiar malady. "In the fall of 1818, the scantily settled region in the vicinity of Pigeon Creek—where the Lincolns were then living—suffered a visitation of that dread disease common in the West in early days, and known in the vernacular of the frontier as the milk-sick! It hovered like a spectre over the Pigeon Creek settlement for over ten years and its fatal visitations and inroads among the Lincolns, Hanks, and Sparrows finally drove that contingent into Illinois. . . . It not only took off the people but it made sad havoc among the cattle. One man testifies that he lost four milk cows and eleven calves in one week. This, in addition to the risk of losing his own life, was enough, he declared, to ruin him, and prompted him to leave for points farther west! . . . . Early in October of the year 1818, Thomas and Betsy Sparrow fell ill of the disease and died within a few days of each other. Thomas Lincoln performed the services of undertaker. . . . . Meanwhile Abe's mother had fallen a victim of the insidious disease. Her sufferings, however, were destined to be of brief duration. Within a week she, too, rested from her labors."

Repeated references in the years that followed are made to milk sickness in historical and medical literature. The rôle of weeds was surmised very early, but in 1861 evidence was produced which tended to confirm the relation of white snake root to the disease in cattle. In 1867 William Jerry of Madison County, Illinois, produced evidence that white snake root could poison human beings. An illness, indistinguishable in its symptoms from what is now known as milk sickness, followed a meal in which white snake root had been eaten as greens. In 1907 Brown (1) called attention to the sweetish odor of the breath and Walsh (2) diagnosed the human disease as an acidosis.

"Alkali disease" had been known in the Pecos Valley of New Mexico and Texas since its earliest settlement.

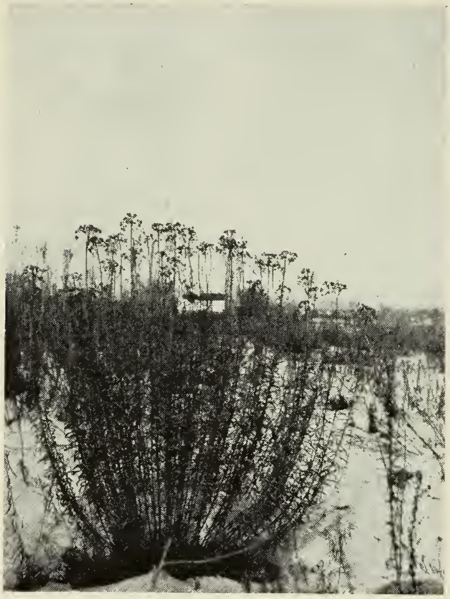
The poisonous properties of the rayless goldenrod were first brought to the attention of the United States Department of Agriculture, when E. O. Wooton (3) of Mesilla Park, New Mexico, submitted a specimen of the plant together with a history of losses in horses and cattle. Since that time a considerable number of reports have been recorded of alkali disease and milk sickness in New Mexico and Texas. The exact nature of the poison responsible for the disease was unknown until recently. In 1909 Moseley (4) published a report in which he ascribed the cause to aluminum salts in the plant. The work of Jordan and Harris (5) indicated that it was of bacterial nature—*Bacillus lactimorbi*—but these findings were not confirmed by other investigators.

In 1926, Couch (6) of the United States Department of Agriculture, isolated from white snake root and the rayless golden rod the chemical substance responsible for the trouble. It has been named trematol.

The Poisonous Plants

Two plants are responsible for milk sickness: white snake root and the rayless goldenrod.

White snake root *(Eupatorium urticaefolium)*, also called richweed, boneset, polewort, or squaw weed, grows from Minnesota to Louisiana and east to North Carolina. There are over forty species of white snake root in the United States, but this is the only one that has given serious trouble. One other member, *Eupatorium serotinum,* has been suspected of causing death in animals, but evidence has not been confirmed. The common thoroughtwort belongs to the same family and grows in the same locality.



U. S. Dept. of Agriculture

FIG. 13.—Rayless Goldenrod.

Eupatorium urticaefolium grows in densely wooded areas. When the trees and underbrush are removed sufficiently to permit the growth of bluegrass the white snake root disappears almost completely. The plants do not thrive in the bright sunlight. It has been known to grow in pastures, however, and is found sparingly over a wide area in urban and rural communities.

In the more highly cultivated agricultural sections it has been found in orchard pastures.

The plant is a slender erect perennial herb which grows from one to four feet high. The leaves are opposite each other, three to five inches long, broadly ovate with sharply toothed or serrated edges. The leaf stalks are about one-fourth to one-half as long as the leaf. Each leaf has three main veins which extend from the base of the leaf and which give off many branches. In the late summer the white flowers of the plant appear as compound clusters having eight to thirty flowers, giving to pastures in heavily infested woodlands an attractive appearance. Other members of the family, including *Eupatorium serotinum,* grow more abundantly in open pastures.

The rayless goldenrod, or jimmy weed *(Aplopappus heterophyllus),* is distributed over a wide territory from southern Colorado to the Texas Panhandle and south to Arizona, Sonora, and Chihuahua. It is especially abundant in the irrigated portions of the Pecos Valley in Texas and New Mexico.

The plant belongs to the composite family, bearing the flowers in compact heads as does the sunflower. It is a stout, erect, tufted perennial herb of one or two feet in height, except on the banks of irrigated ditches where it grows four feet or more high. The leaves are alternate, linelike or broader, one-eighth to one-fourth inches wide, and three-fourths to two and one-half inches long. The heads are numerous, with or without stems in terminal, flat-topped bunches, each head having seven to fifteen tubular flowers.

TREMATOL

Trematol is the name given by James F. Couch of the Bureau of Animal Industry of the United States Department of Agriculture to the poisonous principle found in white snake root responsible for trembles in cattle and milk sickness in man.

White snake root contains three poisonous substances, a complex alcohol, a resinous acid, and a volatile oil. The latter two are only slightly poisonous and do not produce trembles. The complex alcohol is the substance that has been named trematol. It is an optically active alcohol of the formula $C_{16}H_{22}O_3$ and is soluble in alcohol, ether, chloroform, benzene, and other organic solvents. It is insoluble in water, acids, or alkalis.

Trematol may be altered by heat or by chemical reagents and when so changed it loses its poisonous properties. The temperature required for the pasteurization of milk, however, does not affect it.

Couch has devised a test for trematol in plants, in milk, butter, cheese or meat. He describes it as follows:

> "The suspected material is extracted with petroleum ether. Ordinary gasoline may be used if it does not give a red color with sulphuric acid. The solution is poured on the surface of 2 c.c. of concentrated sulphuric acid in a dry test tube, when if trematol is present, a red color appears at the junction of the two layers. Upon shaking the tube the petroleum ether layer is colored a transient red and on allowing the layers to separate the lower layer of acid is colored a fine cherry red while the upper layer is colorless. If the solution is very dilute only an orange color will result."

Samples of materials that fail to give the test are probably innocuous but present knowledge would not justify the statement that such samples were actually harmless. Milk, butter, or meat that respond to the color reaction are dangerous to health and should not be used as food. A few substances other than trematol give a red color with sulphuric acid, but they are not likely to be present when trematol is suspected.

Trematol is rapidly destroyed by drying so that completely dried snake root is incapable of producing trembles. The poison of the rayless goldenrod, on the other hand, is not destroyed by drying and this plant is dangerous to cattle either green or dry.

The Disease in Animals

Trembles appears only in pastured animals—horses, cattle, and sheep being the ones naturally affected. During the autumn when pastures are dry, white snake root is green and cattle often turn to it as a last resort. Sometimes cattle will eat it even though pasture grass is abundant. The course of the disease is practically the same whether due to white snake root or the rayless goldenrod.

The first symptoms in cattle are drooping of head and ears, dull appearance, loss in weight, abnormal thirst, obstinate constipation, and general inactivity. A characteristic trembling of the voluntary muscles is responsible for the name trembles.

Marked muscular debility is noted in advanced stages when the animal lies prostrate on the ground unable to rise. The breath has a marked pungent odor. The pulse and respiration become irregular and slow as coma appears, followed by death. Not all cattle in a herd, even though kept under the same conditions, develop the disease, while the severity of symptoms of those infected varies in different animals.

Milk cows exposed to white snake root may transmit the poison through the milk without showing any symptoms of the disease. It would appear that the milk absorbs the poison thus protecting the animal in lactation. When the animal goes dry the system at once becomes susceptible and the disease runs its usual course described above. Sometimes animals in lactation show symptoms, however.

Excreted
rather into
the milk.

The disease runs a more rapid course in horses than in cattle. The usual symptoms seen in cattle are observed. Paralysis of the throat results in slobbering which is an early symptom. Death follows in two or three days.

Sheep exhibit symptoms similar to those seen in cattle. A drowsy, sleepy condition, bordering on coma usually precedes death.

Hogs are considered immune and may forage on infected ground without danger. Walsh states, however, that hogs fed on animals dead of the disease may develop the disease themselves and die.

Experimental feeding of white snake root was carried out at the Illinois Experiment Station in 1922 (7).

> The plant was fed fresh after being cut and delivered 20 miles by automobile each day. One cow died in 7 days after consuming a total of 12 pounds of the plant on 5 consecutive days. A horse died on the sixth day after consuming a total of 30 pounds for 5 consecutive days. A ewe, nursing a lamb, died on the fourteenth day after eating a pound of white snakeroot daily for 13 consecutive days. The lamb nursing the ewe died on the ninth day. One cow and nursing calf, one steer, and one cow without calf showed no symptoms of illness following the ingestion of the plant for 30 consecutive days. One sow and nursing pigs were fed liberal amounts of the plant for 30 consecutive days without noticeable results. Two goats showed no symptoms after eating the plant for a similar length of time. Fresh milk from cows showing symptoms of trembles induced by feeding the snakeroot was fed to 2 pigs, 1 bitch suckling 4 puppies, a 5 month old lamb, 1 monkey, 4 chickens, 2 cats and 6 guinea pigs in amounts varying from 4 c.c. to 200 c.c., without producing symptoms of white snake root poisoning.
>
> The toxic character of milk from cows 10 days after being pastured on white snake root was only occasionally suggested by the fatal character of thirty drops of the freshly drawn milk when administered subcutaneously to guinea pigs. The sterile milk serum from the same milk samples following filtering produced death in one instance when injected into guinea pigs. Milk samples from cattle feeding on white snake root did not prove consistently toxic, and definite conclusions are not drawn from the few positive findings."

Prevention of trembles is better than attempts at cure. Each case that arises may be treated separately under the care of a veterinarian but cure is not always certain.

THE DISEASE IN MAN

Milk sickness in man is the result of ingesting the poison trematol from milk, beef, butter, or cheese from cattle which have partaken of white snake root or the rayless goldenrod. The poison is cumulative, causing a restless, weak, exhausted, languid feeling during the early stages of the disease. Later abdominal pains develop, with nausea, vomiting, constipation, thirst, loss of appetite, weak pulse, labored breathing, and subnormal temperature. The throat and intestinal tract apparently become paralyzed and in fatal cases death is often preceded by a prolonged coma. The incubation period is two to twelve days. Acetone breath is a characteristic symptom. The fatigue may last a long time with characteristic recurrences. Walsh describes a case poisoned in 1924 and repoisoned again in August, 1925. Three months after the poisoning strenuous exercise brought on a marked attack of the disease.

Couch believes that poisoned animals or man suffer from a ketosis which is a subsequent symptom to the toxic factor responsible for the onset of the disease. Acetone has been demonstrated both in the urine and expired air of the lungs.

The prevalence of the disease in man is difficult to estimate as very few of the actual cases are reported. In Illinois during the summer of 1926 there were newspaper accounts of fifteen deaths but death certificates were turned in by physicians for only seven cases during the year. Since the mortality is considered to be about 25 per cent, there probably occur under normal conditions at least fifty cases of milk sickness in Illinois each year. Conditions in other states where white snake root exists to any degree are not dissimilar. Education of the public has had a marked effect upon human affections. A publicity campaign instituted by the State Department of Agriculture in Illinois resulted in 1927 in only two human cases and no deaths.

Prevention

The prevention of trembles in cattle depends upon avoiding pastures badly infested with white snake root. Cattle, horses, and sheep should be kept out from July 1 to December 1, or until the pastures are cleaned of the weed. This may be accomplished by systematic pulling of the plants in August and September while the flowers are in bloom, and repeated in October. After the plants have been pulled and allowed to dry they should be burned to destroy the seed. Prevention of milk sickness in man depends upon avoiding meat or milk products from affected cattle. A policy of education in Illinois almost entirely eliminated human cases in 1927, while the year before they were more or less prevalent.

Milk intended for human consumption is not rendered safe by pasteurization. Under ordinary market conditions, however, such poisoned milk is diluted sufficiently with milk from other sources to render it harmless. There need be no fear among city dwellers of outbreaks of milk sickness of great magnitude. Milk sickness is a rural problem and education, therefore, must be with the farmer.

Items of Note

1. Milk sickness is due to a chemical poison found in the white snake root plant or the rayless goldenrod. This is in distinction to the other animal diseases which are bacterial in nature.
2. Trematol, a complex alcohol, is the responsible agent in white snake root. The toxic agent in the rayless goldenrod is somewhat different in nature.
3. The poison is of danger chiefly to cattle, horses, and sheep.
4. Man is poisoned from ingesting meat, milk, or milk products from affected cattle.
5. Trematol in milk is not destroyed by pasteurization temperatures.
6. Market milk is ordinarily rendered safe by dilution of the poisoned milk with milk from other sources.
7. Milk sickness in man is largely a rural problem.

BIBLIOGRAPHY

1. Brown, T. R.
Osler's Modern Medicine, Philadelphia, 1907, *3,* 547.
2. Walsh, W. E.
Illinois Medical Jour., 1909, *15,* 422.
3. Marsh, C. D., Roe, G. C., and Clawson, A. B.
U. S. Dept. Agri., Dept. Bull. 1391, 1926.
4. Moseby, E. L.
Med. Rec., 1909, *75,* 839-844.
5. Jordan, E. O., and Harris, N. M.
Jour. Inf. Dis., 1909, *6,* 401-491.
6. Couch, J. F.
U. S. Dept. Agri., Bur. An. Ind., Circular Letter on Milk Sickness, Oct., 1926.
7. University of Illinois
Div. of Animal Pathology, Animal Pathology Exchange, Aug. 1927.

Chapter VI

ACTINOMYCOSIS

Actinomycosis is a chronic infectious disease caused by *Actinomyces bovis* or related members of the ray fungus group. It is characterized by the growth of granulation tumors, which eventually supporate discharging granular bodies characteristic of the infection. While no portion of the body is immune from invasion, the head and neck very often are attacked, which, with the proliferation of the surrounding connective tissue, gives rise to the name in cattle of lumpy jaw, wooden tongue, or sarcoma of the jaw.

History

In 1845 Langenbeck first recognized the "clumps" produced in lesions of this disease. In 1877 Bollinger discovered the organisms, which were given the name "actinomyces" by Harz—a botanist. Cultural and pathogenic studies have been made by Boström (1890), Wolf and Israel (1891), and others. Since the disease is not very contagious it is not of great economic importance, and hence does not figure largely in the literature.

Animals Susceptible

The following animals have been found susceptible to infection with one or another of this group of organisms: Man, cow, horse, pig, sheep, dog, goat, monkey, guinea pig, rabbit, mouse, goose, lizard.

Types of Organisms

The genus *Actinomyces* contains sixty-four members, most of which are harmless saprophytes. *Actinomyces bovis* is responsible for the disease in cattle and for many cases in man; *actinomyces hominis* is closely related but has been found in man only. Stokes found seven species pathogenic for man and animals, all of which produced characteristic changes in tissue. Bergey (1), however, lists ten members of the group as follows:

Actinomyces bovis—the cause of actinomycosis in cattle.
Actinomyces hominis—the cause of actinomycosis in man.
Actinomyces madurae—the cause of madura foot.

Actinomyces freeri—transmissible to monkeys, dogs, and guinea pigs.
Actinomyces actinoides—found in epidemic pneumonia of calves.
Actinomyces gedanensis—isolated from sputum of patient suffering with chronic lung disease.
Actinomyces candidus—from lung in pulmonary tuberculosis.
Actinomyces asteroides—from cerebral abscess in man, and in conditions resembling tuberculosis.
Actinomyces farcinicus—associated with disease in cattle resembling chronic tuberculosis.
Actinomyces necrophorus—cause of "diphtheria" in cattle with multiple sclerotic abscesses.

The organisms grow in the form of a much branched mycelium, which may break up into segments that function as conidia. In lesions these form a ball-shaped network with radiating club-shaped mycelia—hence the name ray-fungus.

Many of the members of this group are anaerobic and are grown with great difficulty in the laboratory. The list of pathogens given above, however, are all aerobic with the exception of *actinoides* and *necrophorus*. At best, new cultures are isolated with some difficulty, growing slowly and resembling the tubercle bacillus on culture media.

The threads are stained with the ordinary dyes. In younger cultures they are from 0.3 to 0.5 microns in diameter, but in older cultures showing branching and club forms, they may be 6 to 8 microns. Often rods and cocci are found in colonies instead of the thread forms. They are non-motile. The spores are very resistant, withstanding drying for months.

Geographical Distribution

Actinomycosis probably occurs in many parts of the world, though figures are lacking. It has been reported from Europe, North America, and South America. In the United States, Sanford and Voelker (2) collected reports upon about 570 human cases from every state in the Union. The Mississippi Valley and the Northwest, however, showed the greatest number of cases.

The Disease in Animals

Actinomycosis runs a chronic course in animals. It may attack any part of the body but infections of the head and neck are most common. The disease is not very contagious. Salmon kept 21 healthy cattle tied alternately between cattle infected with actinomycosis, but no cross infections occurred. Probably, most infections are caused by accidental traumatisms whereby the organisms gain entrance. The cereal grains, which are capable of penetrating the tissues, have been found in the center of actinomytic infections, but Wright denies the presence of the microorganism on such grains.

Cattle are most often affected. A lesion forms at the site of inoculation which takes the form of a tumor-like growth, sometimes resembling that of tuberculosis. This eventually breaks down, discharging thick yellowish pus. Infections around head and neck are accompanied by proliferation of connective tissue. When the tongue is infected it becomes swollen and often protrudes from the mouth—hence the name wooden tongue. Sometimes the bone of the jaw is attacked and the lesion mistaken for osteosarcoma. Lesions of the mammary gland are not common, but, when they do occur, are often mistaken for tuberculosis. Patterson found 5 out of 50 cows suspected of suffering from tuberculosis to be in reality infected with actinomycosis. In each instance the lesion was on the udder, probably infected by straw.

Other animals are less commonly infected than cattle. A few cases have been found in horses, usually with the submaxillary lymphatic glands involved. In the pig, lesions have been found in the mammary glands, throat, and castration wounds. Sheep are infected on the tongue, lips, and in the lungs. Dogs and cats have been found infected, according to several workers. Geese have shown lesions in the pericardial and peritoneal sack. Lizards have been infected by experimental inoculation, as have monkeys and some of the other experimental animals. Experimental inoculation with pure cultures very often ends in failure, however.

The Disease in Man

Actinomycosis in the human subject is principally a disease of males—probably because they are more often exposed and because they may be less careful in their personal habits. Among Sanford's 670 cases in the United States, 80 per cent were in males. While the majority of the cases were among young adults, there was no age group which was immune. The youngest case was an infant twenty-eight days old, described by Stokes in Baltimore, while there were 22 others less than ten years old. Two patients were eighty-two years old.

There is no organ of the body which may not be affected. Sanford found 60 per cent of the lesions were around the head and neck, 18 per cent abdominal, 14 per cent thoracic, while rarer forms involved the pelvis, gluteal region, peritoneal region, scrotum, arms and forearms, hands, fingers, thumb, perineum, foot, heel, thigh, ovary, tubes, kidney, bladder, testis, and penis. Luckett states that actinomycosis of the bone is very rare.

The manner of infection is often in doubt. Cattle may be a source sometimes, but it is probable that infection directly from nature, similar to the mode in which cattle are infected, is the usual procedure. Ewing, who collected records on one hundred cases in the United States in 1902, could find no evidence of infection from animal to man, either through milk or meat or by accidental inoculation. Persons who suck or chew straws, grains, or weeds lay themselves open to infection. The presence of a careous tooth allows entrance of the spores. The cereal grains are capable of penetrating the soft tissues, carrying in the infection. Cutaneous infection has been caused by wood splinters.

The disease may be acute, sub-acute, or very chronic. The shortest symptoms on record are one week while the longest are fifteen years. Ordinarily, however, it is a very long drawn-out disease.

The symptoms may simulate syphilis, tuberculosis, sarcoma, carcinoma, or mycetoma.

Almost all cases recover, but occasionally generalized infection with another organism sets in, resulting fatally.

Prevention

Since most cases of actinomycosis in animals are due to accidental infections from nature, there is little that can be done to prevent them. The disease in man can often be guarded against by abstaining from chewing straws, weeds, and grains.

Items of Note

1. Actinomycosis is only slightly contagious, infection usually arising accidentally from nature.
2. In man the disease is largely rural in character, attacking, in most instances, young adult males.

Table 22

ACTINOMYCOSIS IN THE UNITED STATES

Sanford and Voelker

Locality	Human cases	Years	Animals
Maine	7	1908–1918	Fraction of 1%
New Hampshire	—		Not prevalent but present
Vermont	1	1922	1 case in 20 years
Massachusetts	58	1894–1922	46 cases in 5 years
Rhode Island	2	1917–1921	
Connecticut	6	1912–1917	
New York	67	1888–1922	
Pennsylvania	21	1899–1922	
New Jersey	4	1902–1922	
Delaware	1		
Maryland	12	1895–1919	
Washington, D.C.	1	1901	Florida State Veterinarian stated 87,151 cattle inspections. 40 showed Act. 0.0004%
Virginia	10	1896–1920	
North Carolina	3	1900–1920	
South Carolina	1	1922	
Georgia	1	1918	
Florida	1	1921	
Michigan	21	1907–1922	
Ohio	19	1896–1922	
W. Virginia	2		
Indiana	12	1904–1922	
Kentucky	1	1900	
Illinois	52	1884–1923	
Wisconsin	47	1886–1923	
Minnesota	52	1887–1923	
Iowa	36	1892–1922	
Missouri	18	1906–1922	
North Dakota	45	1923	
South Dakota	23	1909–1923	
Kansas	19	1898–1921	
Nebraska	9	1913–1919	
Tennesee	7	1898–1921	
Arkansas	2		
Alabama	3	1897–1922	
Mississippi	3	1922	
Oklahoma	12	1906–1922	
Louisiana	4	1921–1922	
Texas	6	1916–1921	
Montana	24	1907–1922	
Idaho	—		
Wyoming	3	1919	
Nevada	2	1906	
Utah	—		
Colorado	7	1909–1921	
Arizona	2	1923	
New Mexico	2	1917	
California	17	1893–1921	
Oregon	2	1917–1920	
Washington	8	1908–1921	

3. While the majority of infections are around the head and neck, no part of the body is free from danger.
4. Cattle infected with the disease do not form a menace to man as a source of milk supply provided the udder is not infected, nor as a source of meat provided the infected part is cut away.

BIBLIOGRAPHY

1. Bergey, D. H.
Manual of Determinative Bacteriology, 2nd Edition 1925, p. 347. Williams and Wilkins Co., Baltimore.
2. Sanford and Voelker
Arch. Surg., 1925, *11,* 809.

CHAPTER VII

SMALLPOX AND COWPOX

Variola, or smallpox, is primarily a disease of man; secondarily it infects cattle, and is then called cowpox. From cattle the disease may be passed back again to man in such a modified form that it produces a very mild type of disease, vaccinia, which protects against smallpox infection.

HISTORY

The history of smallpox is too well known to need extended recital. Its beginnings are lost in the annals of time. Its original home cannot be traced, although some authorities believe it may have developed first in certain regions in Asia and Central Africa.

The first exact description of the disease was given by Rhazes, an Arabian physician, in the ninth century, A.D. It had existed for many centuries before the Christian era in China, however. The *"pesta magna"* described by Galen in the second century is believed to have been smallpox. The disease was prevalent in the sixth century and existed again among the crusaders. The ravages of smallpox have been the scourge of peoples for centuries. During the eighteenth century alone 60,000,000 people are estimated to have died from it. It was a disease of childhood, most children contracting it before the age of seven; those few children who escaped were almost certain to become infected later. Numerous references in the poetry and prose of English literature indicate the fear and terror which it inspired in the popular mind. Ben Jonson, about 1600, wrote:

> "Envious and foule disease, could there not be,
> One beautie in an age and free from thee."

Lord Macaulay has given a powerful word picture of conditions about 1700:

> "The smallpox was always present, filling the church yard with corpses, tormenting with constant fears all whom it had not stricken, leaving on those whose lives it spared the hideous traces of its power, turning the babe into a changeling at which the mother shuddered, and making the eyes and cheeks of the bethrothed maiden objects of horror to the lover."

Smallpox was introduced into the Western Hemisphere by the Spaniards about fifteen years after the discovery of America, where it proceeded to render terrific havoc among the non-immune native population. Catlin estimated that of 12,000,000 American Indians, 6,000,000 died of smallpox. Numerous figures from other parts of the world are on record indicating a similar relative importance of smallpox in times gone by.

Under such conditions Jenner was reared. Cowpox was also prevalent in England at that time and it was common gossip that an attack of cowpox would protect against smallpox. A milkmaid at Sudbury remarked in the presence of Jenner that she could not contract smallpox because she had had cowpox. This statement possibly was responsible for initiating the investigations that followed. On May 14, 1796, Jenner collected lymph from the hand of Sarah Nelms, a dairy maid infected with cowpox, and transferred it to the arms of James Phipps, an eight-year-old boy. A typical take followed. Six weeks later the boy was inoculated through incisions on both arms with material from a smallpox pustule, with no take. Experiments were continued until Jenner was absolutely certain of his results. In America the first vaccination with cowpox lymph was made in 1800 by Waterhouse, who vaccinated his five-year-old son with vaccine virus obtained on threads from England. In 1802 a most crucial experiment was carried out in Boston, when nineteen boys were vaccinated with cowpox virus. Three months later twelve of them were inoculated with smallpox, without results, although two unvaccinated boys similarly inoculated with smallpox virus contracted smallpox. Inoculations with smallpox virus were repeated on the vaccinated boys again without results. This experiment did much to establish faith in America in this method of protection against smallpox.

THE ETIOLOGICAL AGENT

? Smallpox and vaccinia are caused by a filterable virus of unknown etiology. Numerous investigators have found peculiar bodies in either the lymph or the lesions which have been considered the etiological agent. Evidence is still lacking, however, concerning the exact status of such bodies.

Guarniéri bodies, described by Guarniéri in 1892 (1), are characteristic structures found in the cornea of rabbits inoculated either with variola or vaccinia material, but never found otherwise. He named the bodies *Cytoryctes variola—vaccinae.* The bodies lie in the cells of the conjunctiva and in the connective-tissue cells of the cornea. These observations have been confirmed by numerous other investigators. The significance of the structures, however, is uncertain, and it is not known whether they are the parasite itself or simply harbor the parasite.

They do not pass Berkefeld filters while both vaccine virus and variola virus do so.

Since they are always present they can be considered pathognomonic of the disease, differentiating it from other infections.

Other structures have been described by different workers. Pfeiffer (2) and van der Loeff (3) saw certain bodies in the blood stream of vaccinated men and animals and of variola patients and considered them the cause of the disease. Paschen (4) as well as von Prowazek (5) and Fornet discovered small round bodies in the lymph and skin lesions of vaccinia in children. Paul (6) inoculated the cornea of rabbits with the bodies, obtaining characteristic reactions. Other investigators have corroborated these findings, especially Scott and Simon (7). The Paschen bodies pass through the Berkefeld filter.

Vaccine virus may be propagated indefinitely by continuous reinoculation of the cornea of rabbits or by inoculation into the testes of rabbits. Recently Parker and Nye (8) cultivated it in tissue cultures of cells taken from rabbits' testes.

The virus is destroyed by heating at 100° C. for three minutes. It is especially resistant to drying, remaining alive for 229 days, according to Paschen. Such disinfectants as phenol and cresol have little effect, the virus withstanding 0.5 per cent phenol for five hours. Noguchi used 1 per cent phenol with no destructive effect. Glycerin in 50 per cent strength seems to exert a selective action, destroying other bacteria but not this virus.

The Relation of Smallpox to Cowpox

Half a dozen diseases characterized by similar pustular eruptions are found among man and animals. Their relation has been the basis of much contention. As far as man is concerned, however, interest centers about the possibility of the unity of variola of man and cowpox of cattle.

Jenner (24) believed that grease of horses, cowpox, and smallpox were modified forms of the same disease. Experimental evidence since that time has accumulated to prove that grease is a separate affection but that cowpox and smallpox are related. According to Ford, as early as 1825, Billing in Stockholm, Brown in England, and Smith in Baltimore reported positive results concerning the identity of cowpox and smallpox. The Lyons Commission (9) in France about 1864 produced vaccinia in cows by inoculation with smallpox material. Voight (10) and Fischer (11) in Germany about 1890 obtained similar results under conditions free from criticism. Freyer (12) not only produced typical vaccinia in cows by the use of smallpox material, but he used the lymph from this animal for inoculation against smallpox with successful results. In England Copeman (13) carried out careful experiments. Smallpox lymph taken directly from a smallpox patient was inoculated into a calf with negative results. It was inoculated onto a monkey with a successful "take," and after one or more passages through monkeys it produced a typical "take" on a calf. Lymph from the calf was glycerinated and used for vaccination of children with successful results. The "takes" were typical and were accompanied by no bad subsequent results. More than a score of other investigators have produced records of successful attempts to modify smallpox into cowpox.

Recently Gordon produced specific antibodies for complement fixation, and agglutinins and lysins, in rabbits by the injection of dead vaccine virus which gave identical results with both vaccine and variola.

It can be assumed therefore that the viruses of *variola vera* and of *variola vaccinia* are the same. When it has been converted into vaccinia by passage through the cow, it has lost its virulence for man and never again produces smallpox.

Cowpox *(variola vaccinia)* as a disease of cattle, originated in all probability from human smallpox, sometime in the past centuries. According to Roseneau, cowpox was much more prevalent in the pre-Jenner days when smallpox was prevalent. Casual cow-pox may be passed from animal to animal in various ways, largely through the hands of the milker. Cows may also be infected with the disease from persons recently vaccinated. Boerner (15) in Pennsylvania reports an outbreak started in such a manner which spread through the entire herd.

Cowpox is chiefly a disease of milch cows. Bulls, oxen and young cattle are less often naturally affected, lending weight to the contention that the hands of the milker are responsible for most cases of cattle infection.

A variety of animals have been shown susceptible to infection with variola and vaccinia. Virus from smallpox cases was transferred to rabbits by Freyer, to monkeys by Park (16) and by Copeman as well as several others, to sheep by Voight, and to horses by the Lyons Commission. Cowpox likewise has been transferred to the horse, ass, sheep, goat, camel, rabbit and monkey.

Levaditi and Nicolau (17) cultivated vaccinia in the brain of rabbits for a year, finding it still potent for man and animals. It gave typical "takes" on rabbits, guinea pigs, rats, mice, heifers and monkeys, but not on cats or chickens.

Horse pox is comparatively rare. The virus infects both man and cattle and may be identical with variola and vaccinia. Some authorities believe it is identical with contagious pustular stomatitis but this is denied by others. Jenner confused the condition with "grease." The latter is a different affection, however, to be distinguished from horse-pox by its non-contagious nature and by the absence of the typical eruption and of buccal lesions. Indeed Jenner often failed to infect cows when he inserted pus from horses heels into scratches made with a lancet on cows teats. He believed that the virus of "grease" was inocuous after pus appeared and that cows had certain seasons when they were resistant to infection.

Sheep-pox *(variola ovina)* is an infection distinct from cowpox. It is a serious affliction among sheep, being quite fatal, but is not transferred to man or other animals, except possibly goats. Vaccination with cow-pox does not protect sheep against it. Goatpox *(variola caprina)* has a mortality of about 2 per cent among goats but is not transferred to other animals. Man is infected upon occasion, according to Hoare. Swine-pox *(variola suilla)* may infect swine, sheep, goats and man. The disease is distinct from cow-pox however.

Other diseases which have been termed "pox" are affections of different natures. Dog-pox *(variola canina)* is a form of distemper. Chicken-pox *(variola gallinarum)*, goose-pox *(variola anserum)* and pigeon-pox *(variola columbarum)* are probably epitheliosis. Andervont (18) in an extended study of the virus of *epithelioma contagiosum* of fowls could find no evidence that it was even closely related to vaccine virus.

Vaccine Virus

The production of vaccine virus is carried on by several biological houses as well as a number of city and state health departments. For interstate commerce it must be produced in accordance with rigid regulations drawn up by the United States Public Health Service.

The method of producing vaccine virus is similar in different laboratories, the same strain of virus being used in nearly all laboratories in the United States. The virus is propagated on rabbits from which calves are inoculated. In some laboratories the virus is "humanized" at intervals, that is, collected from a person which has been inoculated with it, then passed back through the rabbit and the calf.

Calves two to four months old are selected, females in healthy condition being chosen. The hair is clipped all over and shaved on the posterior abdomen and inside the thighs. The skin is thoroughly washed and sterilized on the shaved area and linear incisions about one-fourth inch apart made with a sharp knife. The "seed" collected from a rabbit is then rubbed over the area and into the incisions. The calves are kept under hospital conditions as aseptically as possible. They are fed on milk alone or on hay that has been sterilized in the autoclave. They are made to stand on raised platforms without hay or straw for bedding.

The vaccine pulp is collected about the fifth or sixth day. The entire inoculated area is washed with sterile water and dried with sterile towels. The scabs are picked off and discarded and the pulpy mass collected with a curette into sterile bottles. The calf is immediately killed and examined by a veterinarian for signs of tuberculosis or other infections of the internal organs. If such conditions are found, the pulp is discarded.

The collected pulp contains not only vaccine virus but a number of other bacteria such as the hay bacillus and staphylococci of the skin. There may also be present streptococci and Welch bacilli.

These extraneous organisms are destroyed by the addition of glycerin, 50 per cent and carbolic acid 1.0 per cent, with storage at a temperature of about 5°F. Investigations carried on in the New York City Health Department (19) have shown that brilliant green in a final concentration of 1:10,000 does not affect the vaccine virus but greatly hastens the destruction of other organisms.

Tests for potency and purity are made before the material is used. The vaccine must be free from tetanus bacilli, must not contain more than 50 organisms per vaccination and must give typical "takes" on a rabbit when diluted 1:1,000 up to 1:30,000, as compared with vaccine of known strength.

In years past vaccine virus was dried on bone "points" or used in bulk. Regulations now prohibit any distribution except in sealed capillary glass tubes.

Efficacy of Vaccine Virus

The protective power of vaccine virus has been demonstrated on numerous instances. In Sweden during the twenty-eight years before vaccination (1774-1801) the annual death rate from smallpox per million population averaged 2,050; in the forty years following vaccination, smallpox deaths averaged only 158.

Wherever a determined effort has been made to vaccinate a large part of the population, smallpox has almost disappeared; when laxity begins to prevail smallpox again becomes prevalent. This is strikingly illustrated in England and in the Philippine Islands.

In England and Wales smallpox vaccination was voluntary prior to 1871. In that year vaccination of infants became compulsory. Following are the deaths from smallpox as given by White.

1867-1876	52,218
1877-1886	18,026
1887-1896	5,092
1897-1906	4,761
1907-1916	139

Even without a subsequent vaccination in childhood, smallpox deaths became very infrequent. In 1898, however, a "conscience clause" was introduced into the law which exempted those who should claim conscientious scruples against the practice. It was only necessary for a "crop" of susceptible individuals to grow up therefore, to have smallpox become again prevalent. The figures for 1919 to 1926 taken from League of Nations Health Reports show the following increase in the number of cases.

1919	294 cases
1920	263 cases
1921	315 cases

1922	973 cases
1923	2,485 cases
1924	3,765 cases
1925	5,363 cases
1926	10,155 cases

In the Philippine Islands previous to the American occupation 40,000 deaths occurred annually from smallpox. When the Americans introduced compulsory vaccination the disease disappeared wherever vaccination was applied. In the provinces surrounding Manila the deaths fell from 6,000 annually to not one. For seven years prior to 1914 there was not a single death in Manila from smallpox. Then laxity began to prevail with a consequent increase in cases and deaths. These were confined, in 93 per cent of the instances, to the unvaccinated, however. Only by resumption of compulsory vaccination was the disease again controlled.

In the United States between 30,000 and 60,000 cases of smallpox occur annually. In most states vaccination is voluntary. In Massachusetts children are required to be vaccinated before entering public schools. The average annual case rate per 100,000 population for the years 1921-26 was only 0.3, while for states where such vaccination is not required, it was very high for the same period—California 104.1, Minnesota 112.7, Washington ? State 152.1.

SMALLPOX IN THE UNITED STATES

1921-26

	Cases	Rate per 100,000 population
States in which vaccination is required for school attendance. Population 36,680,000	20,973	9.5
States in which required vaccination is prohibited or restricted. Population 12,123,000	74,271	102.0

Certain criticisms have been advanced against vaccination. One is that it spreads syphilis. This was possible and probable under the old method of transferring scabs directly from arm to arm; under the present procedure of manufacturing vaccine virus it is impossible. Another is that it is responsible for tetanus. In 1903 Rosenau (20) showed that some of the vaccine virus offered for sale in the United States contained tetanus spores. In 1917 McCoy and Bengston (21) demonstrated the presence of tetanus organisms on bone-point scarifiers which were at that time in use for performing vaccinations. Regulations at the present time are sufficiently stringent to prevent the distribution of vaccine virus contaminated with tetanus spores. Armstrong (22) has studied post-vaccination tetanus accurring in thirty-two states in this country between 1914 and 1928. He found that dressings, bunion pads, shields and the like were responsible for the few cases of tetanus that developed (23). Recent methods of vaccination recommended by the United States Public Health Service absolutely avoid all dangers of tetanus infection.

Items of Note

1. While the cow is responsible for the transfer to man of several disease producing organisms, it acts as a buffer against the spread of smallpox.
2. Smallpox virus passed through the cow is rendered non-fatal to man.
3. Vaccine virus has been found efficient in the suppression of smallpox wherever it has been used.
4. Present governmental regulation make vaccine virus a harmless product.

BIBLIOGRAPHY

1. Guarniéri, G.
Arch. per le sc. med., 1892 *26,* 403-424.
2. Pfeiffer, L.
Cor. Bl. de allg. ärztl. Ver. Thuringen, 1891, *20,* 249-266.
3. Van der Leoff, A.
Monatsch. f. prakt. Dermat, 1887, *6,* 189-195.
4. Paschen, E.
Arch. f. Kinderheilkunde, 1908, *47,* 168-173.
5. von Prowazek, S.
Arb. a.d. k. Gsndhsamte, 1905, *22,* 535-556.
Arb. a.d. k. Gsndhsamte, 1906, *23,* 525-541.
Arb. a.d. k. Gsndhsamte, 1907, *26,* 54-58.
6. Paul, G.
Deutsch. Med. Wchschr., 1913, *39,* 2136-2138.
7. Scott, J. M., and Simon, C. E.
Am. Jour. Hyg., 1923, *3,* 401-415.
8. Parker, F., and Nye, R. N.
Am. Jour. Path., 1925, *1,* 325-335.
9. Chauveau, Viennois, and Meynet.
Report of the Lyon Commission, Rev. de Méd. Vét., 1865.

10. Voight, L.
Monatsch. f. prakt. Derm., 1905, *40,* 485-492.
11. Fischer
Munch. med. Wchnschr., 1890, *37,* 735-737.
12. Freyer
Zeitsch. f. Hyg., 1896, *23,* 322-330.
13. Copeman, S. M.
Proc. Royal Soc., London, v. 71, 1902.
Jour. Path. and Bact., May, 1894.
14. Copeman, S. M.
The Nation's Health, Jan., 1926, *8,* 32-33.
15. Boerner, Fred, Jr.
Am. Vet. Med. Jour., 1923, *64,* 93-97.
16. Park, W. H.
Tr. Asso. Am. Phys., 1902, *17,* 217.
17. Levaditi, C., and Nicolau, S.
Comp. rend. Acad. du Sci., 1922, *174,* 249.
18. Andervont, H. B.
Am. Jour. Hyg., 1926, *6,* 719-752.
19. Krumwiede, C., Fielder, F. S., and Watson, T. A.
Jour. Inf. Dis., 1918, *22,* 118-124.
20. Rosenau, M. G.
Hygienic Laboratory Bulletin 16, 1903. U. S. Public Health Service.
21. McCoy, G. W., and Bengston, I. A.
Hygienic Laboratory Bulletin 115, 1918. U. S. Public Health Service.
22. Armstrong, Charles
Jour. Am. Med. Asso., 1928, *90,* 738-740.
23. Armstrong, Charles
Public Health Reports, 1925, *40,* 1351-1357.
24. Jenner, E.
An inquiry into the causes and effects of variolae vaccinae. London, 1798.

Chapter VIII

GLANDERS

Glanders or farcy is primarily an affection of the equine species; secondarily man is infected through contact with diseased animals.

History

The history of glanders is long and interesting. The disease is mentioned in the writings of Hippocrates about 450 B.C. and in those of Aristotle a century later. In the fourth century A.D. Apsyrtus recognized its contagious character and recommended segregation of infected animals. In the fifth century Vegetius gave the first systematic description of the disease. In 1618, Ruini, an Italian anatomist described it as "a general disease and incurable." Solleysel in 1667 believed it could be transmitted through the air. Caspard de Saunier in France in 1734, tracing the infection through harnesses, water troughs and mangers, considered it highly contagious. A few years later (1749) however, LaFosse, senior veterinarian to the King of France, in "A Memior of the Glanders of Horses" described the disease as only local in character and non-contagious.

This view was not generally held at that time, for Vitet in 1773, as well as Volpi, reported it as virulent and contagious. In 1797 Eric Viborg in Denmark considered glanders and farcy to be the same disease caused by a specific virus, very contagious and spread by harnesses, mangers and utensils. Osiander in 1783 was the first to state that glanders could be transmitted to man.

The early part of the nineteenth century is marked by controversies over the spontaneous origin of life and non-infectiousness of disease. The Alfort school of veterinarians in France were strong advocates of this theory in regard to glanders, with most unfortunate results.

In London Coleman, although he transmitted the disease from a horse to a donkey, thought it might arise without contagion. As late as 1860 Percival published a two hundred page monograph on glanders in which he ascribed its cause to miasms arising from bad ventilation, overcrowding, etc.

The infectiousness of glanders was demonstrated meanwhile by several investigators. Travers and Coleman in 1828, and Rayer in 1837 infected horses with material from human cases of glanders, with profound educational results. In 1840 at Alfort, Renault and Bowley discovered likewise that glanders was contagious.

The development of bacteriology in the latter part of the century effectually established the cause of the disease and put an end to the controversy.

Babes in 1881 was the first to observe the glanders organisms in material from lesions. The next year Bouchard, Capitan and Charin in France obtained it in mixed cultures, while Löffler and Schütz in Germany (1) isolated and studied it in pure cultures.

Mallein was discovered by Helman and Kalning in Russia in 1891.

Prevalence and Distribution

Glanders is influenced neither by seasons, by climate nor by geographical conditions. It has been carried into every part of the world by trade and by war. In the Civil War in the United States it was prevalent and increased immediately afterwards in many places. The same was true of the Italo-Austrian War and the Franco-Prussian War. It was carried into Mexico by American army horses in the Mexican War in 1845 and into Cuba in the Spanish War in 1898, where it spread among the native horses. It was carried by British army horses into Egypt, Abyssinia and Afghanistan. After the South African war the whole country was left infected with glanders.

Glanders has been spread by trade also in years past. Although restrictive measures such as quarantine and the mallein test have cut this source of danger to a minimum, there are enough cases that escape to make the infection a menace wherever horses exist. In 1927 the disease was reported from 22 states of the Union and in Quebec, but is fast declining (7).

The Glanders Bacillus

Pfeifferella mallei, previously known as *Bacillus mallei, Bacterium mallei* or the glanders bacillus, is a small slender rod from 0.3 to 0.5 microns thick and 1.5 to 5 microns long. It is not motile nor does it form spores. Ordinary aqueous stains are not very satisfactory but Loefflers alkaline methylene blue gives good results. Irregularity of staining is not uncommon, some of the rods appearing to possess deeply staining granules.

Growth takes place on ordinary culture media, best at body temperature and with the addition of glycerin or blood serum. Potato gives a good result also.

The organism is not very resistant to adverse conditions. A temperature of 55°C. for ten minutes or 75°C. for five minutes will destroy it, as will direct sunlight in a few hours. It will resist drying for not more than two or three weeks ordinarily, but instances are on record of its survival for two or three months. The common disinfectants in the usual strength solutions are effective in ten or fifteen minutes.

The Disease in Animals

Glanders occurs as a natural infection only in horses, mules and donkeys. Other animals may become infected, however, the most susceptible being guinea pigs, cats, dogs, ferrets, moles, and field mice (Park). Sheep, goats, hogs, rabbits, white mice and house mice are much less susceptible while cattle are immune.

The term "glanders" is used to designate either a nasal or pulmonary form of the disease. The nasal type is characterized by rise in temperature, the formation of ulcers upon the nasal mucous membranes which discharge a thin, virulent mucous, and the involvement of the lymphatic glands which become inflamed and supporate, leaving deep open ulcers. The lungs are likewise involved causing rapid and irregular breathing. In the pulmonary form however, sometimes the nasal symptoms are absent, the animal going some months without external signs of disturbed health. Finally, the condition of the animal becomes poor, with definite symptoms of the disease.

Pulmonary glanders is the most important and the most frequent form of the disease. Its latent manifestation in the form of nodules in the lungs and their breaking down are more frequently responsible for the spread of the disease than any of the other forms. The presence of nasal glanders with its characteristic discharge and clinical manifestations naturally offers some definite sign by which the disease may be diagnosed and the infected animal eliminated. The unsuspected latent cases, however, when lung lesions communicate with the bronchi and the bacilli are eliminated through coughing, etc., are the dangerous forms as far as the propagation of the disease is concerned.

The term "farcy" is used to designate the chronic cutaneous form of the disease, characterized by circumscribed swellings on different parts of the body. These often supporate, leaving open ulcers from which is discharged a purulent material. The lymphatics leading from these ulcers become inflamed and from them new "farcy buds" develop.

The acute form may be fatal to horses in one to six weeks, especially young horses. The chronic form, on the other hand, may run on for years, until overwork or other adverse conditions, bring on death.

The prevalence of glanders in horses has been much reduced in recent years. Previously, when horses were more numerous and sanitation more neglected, the disease was a serious problem in large cities. The dwindling number of horses, coupled with the abolishment of the public water trough, renders the exposure to infection much less. More competent veterinary service with improved methods for early diagnosis and better sanitary standards are a further important factor. Finally the activity of federal, state, and municipal authorities have contributed largely to the decrease in the prevalence of the disease. No statistics are available however.

The Disease in Man

"Glanders is the most painful disease from which man can suffer," according to Gaiger (8) who passed through a course of the malady. It is confined largely to veterinarians, hostlers, laboratory workers or others who come into direct contact either with diseased animals or cultures of the organism. Because of occupation, more cases occcur in males than females. Bollinger found only 6 cases in females out of 120 studied and Robins 15 out of 156 studied.

The acute form of the disease is said to be more common in man with the mortality about 60 per cent. Probably, however, many cases are incorrectly diagnosed as other diseases, especially the chronic form of glanders. In Illinois I encountered a case that was admitted to the hospital for pneumonia and the diagnosis of glanders was made several weeks later only after a positive laboratory examination. The symptoms are often very obscure. In Chicago several years ago a case occurred in a laboratory worker who had been handling glanders cultures, but the disease was not suspected till the patient had been ill sometime and was near death.

Man is quite resistant to infection under ordinary conditions. In a road construction camp in Illinois in 1923 nineteen horses died of the disease and some fifty more infected animals were killed. Yet among the 600 or 700 men in camp who had been in more or less close contact with the animals only one case developed. Exceptions to this normal resistance appear in laboratory workers who not infrequently become infected, even though the ordinary precautions of technic are observed.

Glanders in man is usually a cutaneous infection, an abraded skin being required for the passage of the organisms. Possibility of infection by way of the respiratory tract is doubted by many workers. The frequent infection of laboratory workers, however, leads one to believe that probably there are other routes than through wounds, both by ingestion and by inhalation.

Passage of the disease from person to person is possible but unusual. Park cites an instance of infection of a washerwoman infected from contaminated clothing. Under ordinary conditions, however, a case of human glanders is not far removed from an equine infection. Robins (4) found only 15 cases out of 156 in man to be directly associated with other human cases.

The prevalence of human glanders is not great. In the State of Illinois, which ranks in the point of horse population among the leading states of the Union, there has been only one human death from this disease reported to the State Department of Public Health. Two other non-fatal cases appear in the records, while an investigation by Dr. W. C. Nordholz in Chicago uncovered four more in a period of twenty-five years. Without doubt other cases have occurred but the disease is not prevalent in man and is becoming less so with its better control in horses.

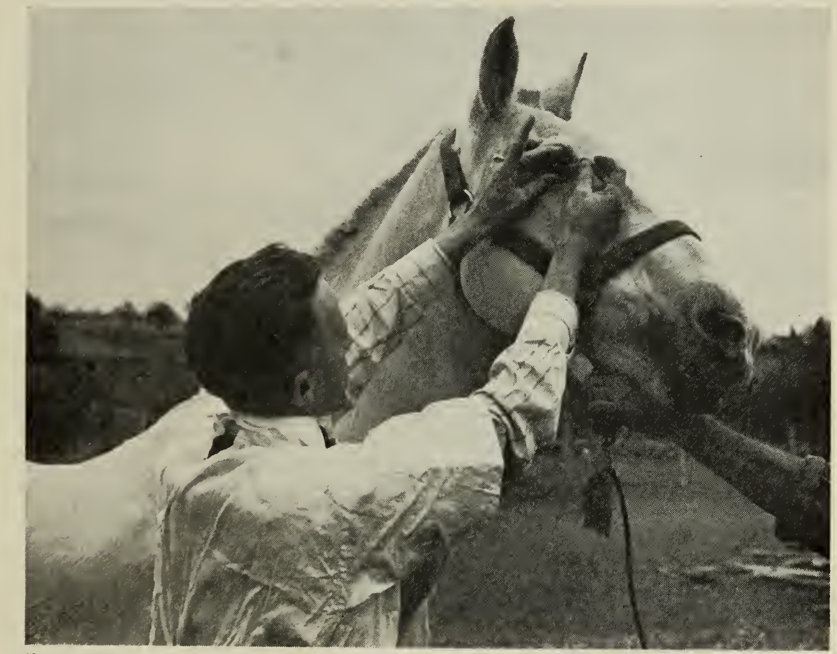
Prevention and Control

The prevention of glanders in man depends upon the education of persons coming in contact with the infection—veterinarians, hostlers, laboratory workers—concerning the dangers of the disease. In the final analysis it depends upon elimination of the disease in horses.

The cure of persons already infected has been attempted with mallein, with vaccines and with immune serum. Robins (2) some years ago reported beneficial results in three out of four patients treated with mallein: in the fourth there was a reaction. The practice has not been generally followed however. Bristow and

White (3) and Cramp (4) each treated a patient with an autogenous vaccine with recovery. There is little evidence on the use of vaccine. Recently Watson (5) produced a potent immune serum in horses by the injection of mallein, using the serum for the cure of three cases of human glanders.

The control of glanders in horses depends upon early diagnosis of cases with immediate destruction of infected animals. Mohler and Eichhorn (6) give four methods for diagnosis of the disease—physical examination, serological tests, mallein reaction and Strauss reaction.



Courtesy of the Lederle Antitoxin Laboratories

FIG. 14.—The ophthalmic mallein test for glanders in a horse.

The physical examination in a case of well developed glanders is sufficient for diagnosis by an experienced veterinarian. Horses in the incipient or early stage of the disease are sometimes more difficult to diagnose. Some horses will carry the infection several months without apparent physical evidence of infection and these are ever present sources of danger.

Serological reactions include both the complement fixation test and the agglutination test. The latter was first applied to glanders by MacFadyean in 1896. According to Anthony and Grund it is reliable in about 84 per cent of positive cases, failing in old chronic cases. In 1909 Schütz and Schubert used the complement fixation test for glanders. It picks up more cases than does the agglutination test, (97 per cent) but fails in the early cases. Utilization of both methods has been found by Huytera and March to give about 99 per cent reliability.

The mallein test is administered in three different ways—subcutaneous, ophthalimic and cutaneous. The material is made after a manner not unlike that for tuberculin, the glanders bacilli being grown in broth which is afterwards filtered, concentrated and glycerinated.

In the eye it produces a severe conjunctivitis if the horse has glanders; under the skin it causes an inflamed edematous swelling with a rise in temperature; on the scarified skin an edema results. Normal horses do not react to the test.

The Strauss reaction is carried out by injecting a small amount of suspected materially intraperitoneally into a male guinea pig. Two to four days later a severe orchitis results if glanders bacilli are present, the testes becoming large and tender, with the skin above them red and shiny. The contents of the testes should be examined for glanders bacilli. A few other organisms will cause the same reaction but according to Buchanan they are all Gram positive. The reaction fails in my experience, in about fifty per cent of the animals. Guinea pigs should therefore always be injected in duplicate.

Items of Note

1. Glanders is primarily an affection of the equine species, secondarily of man.
2. The disease has a world-wide distribution.
3. It has been decreasing in horses in the past few years.
4. Veterinarians, hostlers and laboratory workers are liable to become infected.
5. Infection usually takes place through the broken skin, though infection by ingestion or inhalation may be possible.
6. The disease in man is of relatively infrequent occurrence.
7. Control and prevention depends largely upon eradication of the disease in horses.

BIBLIOGRAPHY

1. Löffler and Schütz
   Deutsche Med. Wochenschr, 1883, *9,* 197.
2. Robins, G. O.
   Studies from Royal Victoria Hospital, Montreal, 1906, v. 2.
3. Bristow, A. T., and White, B.
   New York State Jour. Med., 1910, p. 236.
4. Cramp, W. C.
   Jour. Am. Med. Asso., 1911, *56,* 1379.
5. Watson, E. A.
   Jour. Am. Vet. Med. Asso., 1926, *64,* 146-153.
6. Mohler, J. R. and Eichhorn, A.
   Circ. 191, Bur. Animal Ind., 1912, U. S. Dept. Agri.
7. Report of Committee on Miscellaneous Transmissable Diseases
   Jour. Am. Vet. Asso., 1928, *72,* 967-70.
8. Gaiger, S. H.
   Jour. Comp. Path. and Ther., 1913, *26,* 223.

Chapter IX

RABIES

Rabies is an infectious disease caused by a virus the nature of which is not entirely understood, but which seems to have an affinity for the central nervous system. Primarily it is an affection of the canine race, especially dogs, but it may affect any of the warm blooded animals; secondarily it is a disease of man transmitted usually by the bite of a rabid dog. The term hydrophobia (from the Greek "fear of water") may be applied to the disease in man but it is not true of the disease in dogs. The term rabies (from the Latin "to rave") may designate the disease in any animal, but there are times when it is not descriptive, as in dumb rabies. In the Southern United States the term "black tongue" has been used because the tongue protrudes, becomes congested and covered with dirt.

History

Rabies is a disease of great antiquity, in fact, one of the oldest known diseases of animals. Plutarch asserted that, according to Athenodorus, it was first observed in mankind in the days of the Asculapiadae, the descendants of Aesculapius, the god of medicine. Whitmore relates that in the thirteenth century B.C. Akteon, son of Aristeus, died of the disease. In the fourth century B.C. Aristotle stated "dogs suffer from madness which puts them in a state of fury, and all animals which they bite when in this condition become also attacked with madness." Man was considered immune, however, at that time.

Celsus in the first century A.D. was the first to give a good description of the infection in man, using the term hydrophobia. He states that the disease is caused by a bite of a rabid animal and the wound must be thoroughly washed in water and burned with a hot iron in order to prevent the development of the disease, for after symptoms develop there is no cure and death always follows. Park believes that probably Celsus obtained his information from earlier writings which have since been lost.

Celius Aurelianus in the second century A.D. gave a very good account of the disease in man.

> "In the beginning of the attack, anxiety with no reason for it, irritability and malaise, restless movements, sleep light and disturbed, insomnia, stretching and continual gaping, and an unremitting desire to vomit, an unusual susceptibility to air, no matter how quietly the patient may have been resting, intoleration and loathing of liquids, little desire to drink. When the disease is established there is thirst and at the same time dread of water, at first at the sight of it, later of the very sound or mention of it. This fear extends to oily preparations. The pulse is small, hard and irregular; in certain cases there is a light fever. There are eructations; heaviness of the limbs. The diaphragm is displaced upward and the functions of the intestines suppressed. Urination becomes frequent and of small amounts. There is nervous spasm and trembling. The voice is hoarse like the bark of a dog; breathing is difficult, the body drawn up. Generalized convulsions are occasioned by the entry of persons for fear that they may be bringing water. The face and eyes are congested, the body emaciated. Pallor and sweating of the upper parts. Frequent erections and seminal emissions. The tongue is protruded, there is hiccough, bilious vomiting, frequently black, fear of drink, the hands being held before the eyes on the approach of the drinking vessel, etc." (Stimson).

Other writers of that period and in the centuries that followed mentioned rabies without adding to the knowledge of the disease. During the middle ages reference to it seemed to have disappeared from medical writings, although it is mentioned in other sources. In 1271 it is related that in Franconia thirty persons perished from bites inflicted by rabid wolves, which invaded the towns attacking people, flocks and herds. Likewise in Paris in 1604 an epidemic of rabies prevailed.

Toward the end of the eighteenth century began a period of observation which led up to the work of Pasteur a hundred years later. In 1770 Van Swieten recognized the paralytic form of rabies in man. In 1804 Zinke demonstrated the virulence of saliva from a rabid dog by rubbing it onto the fresh wounds of rabitts, dogs and chickens, all of which developed the disease. In 1813 Gruner recommended the inoculation of saliva from suspected dogs into test animals for the purpose of diagnosis. In 1821 Magendie and Breschet identified human rabies with the disease in animals by infecting a dog with saliva from a human case. In 1829 Hertwig showed that the usual method of infection was the saliva of rabid dogs transmitted by bites. All of these observations were corroborated by other investigators.

During this period of renewed scientific activity after a lapse of nearly two thousand years, it is only natural that false ideas and inaccurate observations should confuse the people. For instance, Morgagni and Zwinger, among others, believed the bite of a dog not suffering from rabies might initiate the disease. Some of the causes ascribed to such spontaneous origin were ungratified sexual instinct in male dogs, sudden suppression of lactation in female dogs, extremes of temperatures, especially of heat, drinking foul water, eating putrified materials or the deprivation of meat from dogs which had been accustomed to a meat diet. Contrasted to these notions were those brought forward by Bosquillon in 1802, that hydrophobia was due to fright and that belief in an infectious agent was a chimera.

It is difficult to realize the hysteria of the populace in those times in connection with rabies. Shooting, strangling, poisoning or suffocating were not uncommon deaths meted out to victims who were even suspected of exposure to the disease. In 1810 a bill was presented in Germany to the affect that "it is forbidden, under pain of death, to strangle, suffocate, bleed to death, or in any other way murder individuals suffering from rabies." As late as 1819 it was related in France that an unfortunate victim of rabies was smothered to death between two mattresses.

In the midst of such confused and hazy notions, and opposed by many persons in high stations, Pasteur began his work in 1880 on the spread and control of rabies. He was able to demonstrate at will the transfer of the virus from animal to animal. He showed that dogs protected by virus attenuated by drying, were refractory to the disease. On July 6, 1885, after great hesitancy, but urged on by the knowledge that the victim would surely perish otherwise, Pasteur administered the first human inoculation of anti-rabic vaccine to Joseph Meister, a nine year old Alsace boy. This was the beginning of a new era in the control of rabies.

Diagnosis of rabies in dogs had been carried out by Pasteur as well as by many others through subdural inoculation of brain material into the rabbit. This process required two or three weeks or more, making it of small value to any person exposed to the disease. Wilson, working in the New York City Health Department Laboratory reduced the period to nine days by use of guinea pigs. It was not until 1903 however that Negri described the bodies in the brain cells of rabid animals. Numerous other investigators soon confirmed his observations that such bodies were diagnostic of rabies.

Seasonal Prevalence

The fallacy that rabies is a summer disease is based upon the fact that during the months of July and August the dog-star Sirius rises with the sun. In ancient times this was attributed as the cause of dogs going mad, hence "dog-days." However, during this period dogs have much greater freedom than in the inclement weather of winter, thus more opportunity for contact with each other. In reality there is not a distinct increase of rabies during the summer months; due to the hot weather very often dogs become cross and are suspected of rabies when they are not infected.

In 1910 Stimson (1) tabulated figures from different parts of the world to show that there was little variation in seasonal prevalence, except that the last quarter of the year showed relatively fewer cases—January to March, 25 per cent; April to June, 27.4 per cent; July to September, 25.2 per cent; and October to December, 21.3 per cent of all cases.

In my own figures collected from various sources, it would seem that the spring months often show the greatest number of cases, with the peak in March (2). The Hygienic Laboratory in a report covering the years 1909-1919 for the District of Columbia, showed this to be the case as well as reports from Kansas and New Jersey for the year 1926. In the State of Illinois the peak seemed to be delayed till June and July, but August and September were the months of low incidence in all cases. There was then a gradual rise through the fall and winter months to the peak in the spring or early summer.

Geographical Prevalence

Rabies knows no geographical boundaries, nor is it subject to terrestrial or climatic influences. There are only a few portions of the globe where the disease does not exist and the only reason for its absence in those places is eternal vigilence in keeping it out. From the frigid vastnesses of the arctic region to the sultry atmosphere of the tropics it has been reported, from the congested quarters of New York and Chicago and London and Paris to the sparsely settled wilds of Russia. Under ordinary conditions the amount of rabies is directly proportional to the population in a given district. In the sparsely settled regions the prevalence is not as great, except where the wild animals become infected.

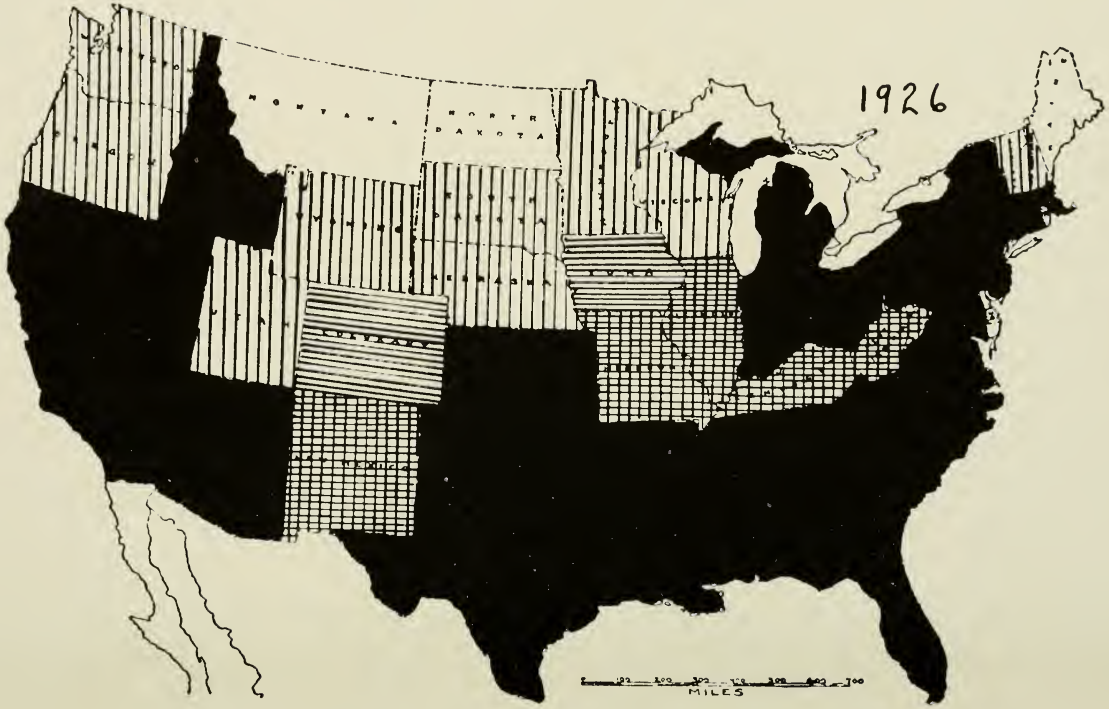

1926
MONTANA
NORTH DAKOTA
NEW MEXICO
MILES

FIG. 15.—Distribution of rabies in the United States in 1926 (*Rice*).

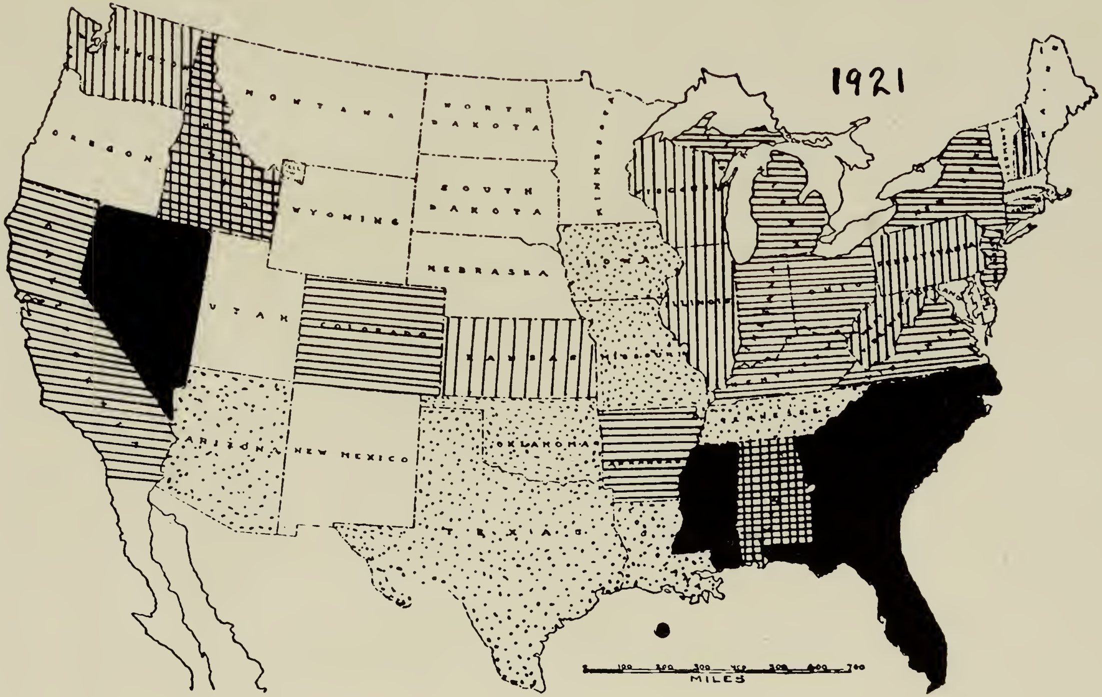

1921
MONTANA
NORTH DAKOTA
SOUTH DAKOTA
OREGON
WYOMING
NEBRASKA
UTAH
COLORADO
KANSAS
OKLAHOMA
ARIZONA
NEW MEXICO
TEXAS
100 200 300 400 500 600 700
MILES

FIG. 16.—Distribution of rabies in the United States in 1921 (*Sellers*).

In parts of Russia rabies among wolves has existed to a considerable extent; likewise in the mountain regions of some of the western states in the United States the disease among coyotes and other wild animals has made its eradication difficult. On the other hand rabies exists to a large extent in the thickly settled portions of the world, unless definite measures are taken to combat its spread.

In 1926 Rice and Beatty (3) published statistics on the world prevalence of rabies which would tend to show very little difference in twenty years in geographical prevalence.

Alberta—No record of any case in ten years.
British Columbia—Only one case in any animal known, and this was in a dog brought in by a tourist. No human deaths.
Manitoba—Some rabies in animals. No human deaths.
New Brunswick—None since 1918. No records previous to that date.
Nova Scotia—None in animals or man.
Ontario—After several years freedom from the disease it was introduced from Quebec and quickly spread. 41 positive brains were examined in 1926. No human deaths.
Prince Edward Island—No data.
Quebec—Rabies spreading rapidly from two foci. No human deaths.
Saskatchewan—No rabies 1926.
Alaska—Not common but has been observed particularly in wolves and foxes.
Arctic Regions—Nansen mentions the occurrence of rabies in "Farthest North" and it has been reported from Greenland.
Mexico—Rabies is very common. There are many Pasteur Institutes and thousands of treatments are given annually. The number of human deaths is rather large but exact information is not available.
South America.
  Ecuador—Not common.
  Venezuela—Rare in animals and extremely so in human beings.
  Argentina—Fairly prevalent.
  Brazil—Has been epidemic at different times especially in the north.
  From incomplete returns it is reasonable to conclude that the disease is present in moderate amount in the greater portion of the continent of South America.
Cuba—Has been epidemic to a serious degree in recent years.
  El Salvadore—Present.
Europe
  Belgium—Has had very little rabies except during the great war when anti-rabic precautions were relaxed. During times of peace elaborate precautions are taken to prevent its introduction. These methods have been highly effective.
  England—By strict muzzling ordinances England was able entirely to rid the country of the disease in 1902. After that time there

was none until 1918 when the disease was re-introduced. It spread rapidly but has again come under control and there has been none since 1922.

France—The home land of Pasteur has long been seriously plagued with rabies. In 1913 it was almost eradicated but flared up again during and after the Great War to alarming proportions. It is at present declining.

Germany—The rabies situation was well in hand before the war there being on the average 300 to 400 cases in animals anually. In 1915, it rose to 1,018, after which there was an irregular increase to 2,699 in 1924. Since that date the condition has improved and is greatly reduced in 1927. Human deaths were as follows: 1924, 48; 1925, 20; 1926, 22. Persons bitten by rabid dogs for the same years were 2,417, 1,159, and 579, respectively.

Holland—Because of very rigid control measures there were no cases of rabies in Holland previous to the Great War. During that crisis there was some in the borders of the country but again none since.

Ireland—There has been no rabies for twenty years. All immigrant dogs are quarantined for six months.

Russia—No communication was received from this country, but it is well known that the rabies situation is very serious, perhaps the worst of any country in the world. The disorganized state of the government, the prevailing ignorance, and the vast territory involved makes the situation well nigh hopeless.

Sweden—There has been no rabies since 1886 when there was one case in an imported animal. Strict regulations govern the importation of all members of the dog family.

Switzerland—Rabies is rare. 329 Pasteur treatments were given in the quarter of a century just closed, and there was one human death.

Austria—Has been epidemic in recent years.

Italy—No very recent data, but has been quite common for a long time.

Balkan States—Has been an important problem here since the Great War and the years immediately preceding.

Spain and Portugal—Rabies very important public health problem.

Africa

Egypt—Very common among the countless homeless dogs. Little effective control of the situation. The number of cases treated trebled from 1914 to 1924, but has decreased since. 1,251 treatments were given in 1926 in Cairo alone.

Union of South Africa—Fairly common in animals and occasional cases in human beings.

Occidental French Africa—Rabies present but not prevalent.

Western Africa—Rabies is common.

Asia

Palestine—Rabies is very common.

Arabia—Common in the settled portions.

India—Data from the Pasteur Institute at Calcutta for the province of Bengal (population, 40,000,000); 1924 (seven months) 1,995 patients treated of whom 17 died; 1925, 5,585 cases treated of whom 33 died. Total deaths for Bengal (human) 304 in 1925.

Japan—A very serious public health problem.

China—Rabies was known in the time of Confucius and has been a serious problem continuously to this day.

Siam—Very common.

Siberia—Very prevalent.

Tibet—Prevalent.

Oceanic Islands

Australia—Free of rabies and always has been so.

Hawaii—Free of rabies. All animals from foreign ports are quarantined for 128 days.

Philippines—Rabies is very common among the many dogs. Human deaths were 103 in 1925; 93 in 1926.

Dutch East Indies—Fairly common.

The Infectious Agent

The nature of the infectious agent of rabies is not entirely understood. Under some conditions it can be passed through a Berkefeld filter, thus placing it among the filterable viruses. By centrifuging, the virus may be thrown down, leaving the supernatant liquid free of infectious material.

The virus is found in the nerve tissue, saliva, urine, lymph milk and other body fluids of infected animals. It may be present in the saliva five days before the animal shows symptoms. Virus obtained from naturally infected animals is designated as "street virus." When the virus has been passed through a large number of rabbits, however, its virulence is so exalted that it will kill the animals in as short a time as six days and this marked virulence is not lost. Such material is known as "fixed virus" (virus fixé of Pasteur).

Rabies virus is not very resistant to unfavorable environment. Sunlight destroys it rapidly while exposure to air and drying soon renders it inert. Boiling immediately destroys it but a temperature of 58° C. requires 30 minutes. The ordinary disinfectants require more time for its destruction than for bacteria—30 minutes for phenol 5 per cent; 15 minutes for mercury bichloride 1:1000, liquor cresolis 3 per cent and formalin 1 per cent. Glycerin serves as an excellent preservative, the virus remaining active for weeks or months in it. Putrefaction does not necessarily destroy the virus for a considerable period of time, but usually putrefied brain tissue is difficult material in which to demonstrate Negri bodies.

In 1903 the Italian investigator Negri, working at Parvia, described certain characteristic bodies in the brain cells of dogs dead of rabies. The large ganglion cells of the hippocampus major and the Purkinje cells of the cerebellum were especially apt to show them. The bodies were round or oval in shape, varying in size from 0.5 microns to 22 microns, the larger ones showing distinct granules when properly stained. Negri considered the bodies not only diagnostic of rabies but the cause of the disease. The presence of the bodies in the brain cells of rabid animals was soon confirmed by numerous investigators in Europe and America and thus the well known method of laboratory diagnosis of rabies was established.

The exact nature of the Negri bodies and their relation to the disease has been the subject of much controversy. Investigators are divided into two groups. The first group agrees with Negri that the body is actually a living parasite, probably protozoan in nature, and is the cause of rabies. The second group holds to the opinion that the bodies are products of cell degeneration, characteristic of the disease, but not causative agents.

The arguments of the second group against the theory of the living parasite are that the virus may be passed through clay filters too small for the size of the Negri bodies seen under the microscope, that the bodies are not found in certain nerve tissues demonstrated otherwise to be infectious and further that the bodies are extremely scarce and of minute size in "fixed-virus" infections.

The first group, contending that the Negri bodies are of etiological significance, point out that the bodies are exceedingly variable in size and it is possible in one stage of the life cycle that they exist in a filterable form. The bodies are also of such definite outlines and structure and stain in such a characteristic manner, that they cannot be degenerative bodies, especially since they may be found outside the nerve cells. It is also possible, by careful trituration, to remove numerous Negri bodies from ganglion cells. In the usual "smear method" of laboratory diagnosis as many Negri bodies are found without the cells as within. Such findings would probably not be possible with degenerated cells.

The protozoan theory has received numerous confirmations. In 1906 Williams and Lowden (4) observed certain cyclic changes in the Negri bodies which made them believe that they were actually dealing with a sporozoan which was the cause of the disease. They designated it "Neurocytes hydrophobiae." Calkins (5) in the same year confirmed the work. In 1913 Noguchi (6) claimed to have cultivated an organism of a protozoan nature, resembling Negri bodies, but his results have never been duplicated by other investigators.

In 1924 Kelser (7) inoculated subdurally a series of rabbits with "fixed" virus. An animal was killed each day, commencing 24 hours subsequent to the inoculation, and the brain examined. After 72 hours structures showing the characteristics of Negri bodies were observed, some as large as those found in "street virus" infection, others apparently dividing by fission and in addition numerous, highly refractile bodies of a minute size. This was several days before symptoms of rabies could be expected. If however, the animal was allowed to develop symptoms and die, only a few, minute Negri bodies were found, indicating that "fixed virus" consists of ultramicroscopic or barely visible forms, which when introduced into the brain of a rabbit, attain a larger size, and undergo cyclic changes. The progeny of such development are mostly ultramicroscopic. It is believed that as a result of a high degree of virulence possessed by "fixed" virus, the disease runs a brief course and the rabbits die before the Negri bodies have had time to attain the size of those observed in the more protracted form of "street virus" infection.

In 1926 Levaditi, Nicolau, and Schoen (8) published an interesting study on the nature of the Negri bodies. After pointing to the tinctorial and morphological similarities between the Negri bodies and the cysts of certain microsporidia, the theory is developed that the Negri bodies represent a stage—pansporoblasts—in the life-history of a protozoal parasite, the units of which at other times in its life history are extremely small and capable of traversing bacterial filters. It is in this spore stage that the virus is eliminated by the salivary glands of a rabid animal and infects the bitten subject. The parasite reaches the central nervous system by means of the nerve trunks and proliferates within the cells of the brain. It must be conceived that all portions of the central nervous system become infected, since all are virulent, but that the development of the organisms is more favored in certain sites than in others; thus the medulla and pons are more intensely invaded and show the infection at an earlier stage than do other parts of the brain, and their cells, moreover, undergo histological degeneration; whereas the cortex cerebri, Purkinje cells and the cells of the cornu ammonis, though infected, retain their histological integrity and show little or no degeneration. These are the very sites in which the Negri bodies develop, an apparent paradox which is explained by Levaditi and his colleagues by the assumption that it is only in the more resistant cells, whose morphological and physiological integrity is preserved for the longest time that the pansporoblasts are formed, only the vegetative phase of the organism existing where its destructive effects are greater.

Schoening (9) in 1925, working in the Bureau of Animal Industry of the United States Department of Agriculture, encountered a strain of "street virus" apparently immunologically distinct from other viruses in that ordinary antirabic vaccines did not protect dogs against it. Kelser believes that the difference is more in variation of virulence than in a distinct type of virus.

Rabies Vaccine

The preparation of all antirabic vaccine is based upon Pasteur's original method—namely the use of nerve tissue from animals which have been infected with the disease, the virus in the tissue being so attenuated or killed that the material is safe for human or animal injections.

Pasteur made the discovery that the fresh spinal cord of a rabbit dead of rabies when emulsified and injected subdurally into another animal, would reproduce the disease in the minimum period—six days for fixed virus. When the spinal cord was allowed to dry for five days a longer period after subdural injection of the emulsion would be required—eight days. A cord dried nine days would require fifteen days to produce rabies, while a two weeks' cord would produce no symptoms at all.

The Pasteur method of antirabic vaccination is based on this phenomenon. Starting with an emulsification of an avirulent cord, sub-cutaneous injections are made on consecutive days, each day using a cord a little more virulent. In intensive treatments of persons badly bitten some of the injections given consist of cord that has been dried only one day.

The original Pasteur schedule has been modified from time to time so that at present at the Pasteur Institute in Paris treatments commence with a five day cord extending over a period of fifteen days for ordinary wounds, with an additional ten days for wounds on the head. The same strain of virus, first used in 1888, was still being used in 1927 after 1,314 animal passages (18).

The Hygienic Laboratory method of the United States Public Health Service begins with two injections of a six day cord, extending over a period of fifteen days for ordinary wounds, with three additional days for severe wounds and six additional for wounds on the head.

Table 23

PASTEUR'S ORIGINAL SCHEDULE OF ANTI-RABIC VACCINATION

Light Treatment			Intensive Treatment		
Day of Treatment	Age of dried cord	Amount of injected emulsion	Day of Treatment	Age of dried cord	Amount of injected emulsion
	Days	c.c.		Days	c.c.
	14	3		14	3
First	13	3	First	13	3
	12	3		12	3
Second	11	3		11	3
	10	3		10	3
Third	9	3		9	3
	8	3	Second	8	3
Fourth	7	3		7	3
	6	2		6	2
Fifth	6	2	Third	6	2
Sixth	5	2	Fourth	5	2
Seventh	5	2	Fifth	5	2
Eighth	4	2	Sixth	4	2
Ninth	3	1	Seventh	3	1
Tenth	5	2	Eighth	4	2
Eleventh	5	2	Ninth	3	1
Twelfth	4	2	Tenth	5	2
Thirteenth	4	2	Eleventh	5	2
Fourteenth	3	2	Twelfth	4	2
Fifteenth	3	2	Thirteenth	4	2
Sixteenth	5	2	Fourteenth	3	2
Seventeenth	4	2	Fifteenth	3	2
Eighteenth	3	2	Sixteenth	5	2
			Seventeenth	4	2
			Eighteenth	3	2
			Nineteenth	5	2
			Twentieth	4	2
			Twenty-first	3	2

The Högyes modification is based upon observations made by Ferran in Barcelona in 1888, who used fresh fixed virus, diluted 1:100 for treating persons bitten by rabid dogs. Boreggi in 1889 was not as successful, losing five patients, apparently infected by the vaccine. Many workers since that time, however, have demonstrated that fresh fixed virus injected subcutaneously, is harmless. In 1897 Högyes began its use, since when more than 100,000 persons have received it without harm. It consists of injections of fresh fixed virus, highly diluted, followed by gradually increasing amounts of the virus. Högyes maintained that the Pasteur method of drying the cord attained nothing more than a dilution of the virus and that this could be more accurately controlled by dilutions with salt solution. Another advantage brought out by Harvey and McKendrick was the smaller amount of nerve tissue in proportion to the amount of virus. The paralysis sometimes encountered during antirabic treatments is said to be due to the large amount of such nerve tissue in the dried cord preparations.

Table 24

SCHEDULE OF ANTI-RABIC VACCINATION AT PASTEUR INSTITUTE, PARIS
In 1927 (18)

Day of Treatment	Age of Cord	Amount injected	
First day	5	3 c.c.	For slight wounds
Second	5	"	
Third	4	"	
Fourth	4	"	
Fifth	3	"	
Sixth	3	"	
Seventh	4	"	
Eighth	3	"	
Ninth	2	"	
Tenth	4	"	
Eleventh	3	"	
Twelfth	2	"	
Thirteenth	3	"	
Fourteenth	3	"	
Fifteenth	2	"	
Sixteenth	4	"	For multiple wounds
Seventeenth	3	"	
Eighteenth	2	"	
Nineteenth	3	"	For serious wounds
Twentieth	3	"	
Twenty-first	2	"	
Twenty-second	3	"	
Twenty-third	3	"	For wounds on the head
Twenty-fourth	2	"	
Twenty-fifth	2	"	

Table 25

PERSONS VACCINATED AGAINST RABIES, WITH MORTALITY, AT PASTEUR INSTITUTE, PARIS (18)

Year	Persons Treated	Mortality	Percent Mortality
1886	2671	25	0 .94
1887	2770	14	.79
1888	1622	9	.55
1889	1830	7	.38
1890	1540	5	.32
1891	1559	4	.25
1892	1790	4	.22
1893	1648	6	.36
1894	1387	7	.50
1895	1520	5	.38
1896	1308	4	.30
1897	1529	6	.39
1898	1465	3	.20
1899	1614	4	.25
1900	1420	4	.28
1901	1321	5	.38
1902	1005	2	.18
1903	628	2	.32
1904	755	3	.39
1905	721	3	.41
1906	722	1	.13
1907	786	3	.38
1908	524	1	.19
1909	467	1	.21
1910	401	0	.00
1911	341	1	.29
1912	395	0	.00
1913	330	0	.00
1914	373	0	.00
1915	654	1	.15
1916	1388	3	.21
1917	1543	4	.26
1918	1803	3	.16
1919	1813	3	.16
1920	1126	6	.53
1921	998	1	.10
1922	754	0	.00
1923	727	0	.00
1924	764	1	.14
1925	782	0	.00
1926	634	0	.00
1927	639	0	.00

The Harris method has been gaining in popularity. The virus cord and brain together are frozen with carbon dioxide snow and finely ground up. The material is then dessicated over sulphuric acid in a vacuum at a temperature of 18° C. In this condition, the powder is sealed up in tubes in vacuo in the proper amounts for treatment and stored below the freezing point ready for use. The powder is emulsified in salt solution when injected. The amounts of material gradually increase as the injections proceed. The method is economical in that large amounts of material can be prepared at once and it will remain potent for six months. Ordinary treatments require ten injections while serious cases take fourteen.

The Cumming method consists in attenuation of the virus by dialysis in distilled water. Emulsified nerve tissue is dialyzed 24 hours in a collodian sac, after which time the material left in the sac no longer is capable of producing rabies when injected subdurally into rabbits, but still retains its protective value. It is administered in amounts of 2 c.c. over a period of 15 to 25 days.

The Semple method has been used to some extent. The brain and cord are emulsified together, diluted to 8 per cent and the virus killed with 1.0 per cent carbolic acid. For use this is diluted half. Fourteen injections are given for human treatments.

The one-injection method for vaccinating dogs also makes use of a fresh fixed virus killed with carbolic acid. The material is so diluted that one injection of 5 c.c. contains 1.0 gram of the original dog brain.

Various other schemes have been tried for the preparation of antirabic vaccine,—altering the virus by heat, by bile and by chemical agents. None of them have prove satisfactory however.

Table 26

ANIMALS SICKENED AND DIED, OR KILLED ON ACCOUNT OF RABIES IN PRUSSIA (1)

Animals	Doebert's figures, 1902–1907		Schüder's figures, 1886	
	Number	Percentage	Number	Percentage
Dogs	3715	85.1		80.2
Cattle	467	10.7		14.7
Horses	63	1.44		1.0
Swine	49	1.12		1.6
Cats	35	.81		.7
Sheep	32	.7		1.7
Goats	4	.09		.1
Other animals				.02
	4365		11,306	

BITING ANIMALS RABID OR SUPPOSEDLY RABID, IN CASES FROM PARIS AND BUDAPEST INSTITUTES

Animals	Paris percentages	Budapest percentages	Animals	Paris percentages	Budapest percentages
Dogs	93.13	90.32	Man	0.04	0.26
Cats	5.75	7.80	Jackals	.03	
Cattle	.37	.52	Sheep	.02	
Horses	.22	.28	Foxes	.02	.02
Asses and mules	.18	.06	Rabbits		.02
Wolves	.12	.38			
Swine	.07	.32	Total number of cases	14,296	4,961

Animals Susceptible

Highly susceptible—all warm blooded animals including man.

Somewhat susceptible—birds and fowls.

Probably not susceptible—cold blooded animals, frogs, turtles, and the like.

Rabies in Animals

Rabies is primarily a disease of the canine race—dogs, wolves, coyotes, etc.—but it may be transmitted to any of the warm blooded animals including man. The lessened prevalence in certain animals may be the result of the relatively infrequent contact with other species which might harbor infection, or possibly because

Table 27

COMPARATIVE REPORT ON 825 ANIMAL HEADS RECEIVED AT ILLINOIS STATE PUBLIC HEALTH LABORATORY 1924–1927

Animals	Total Received	Number Examined	Number Positive
Dogs	731	644	393
Cats	56	50	18
Cows	17	17	2
Skunks	3	2	2
Wolves	2	2	1
Hogs	4	4	0
Sheep	2	2	0
Mules	2	2	0
Horses	3	3	0
Foxes	1	1	0
Monkeys	1	1	0

of natural protection like a thick fur which would mechanically remove the virus that would otherwise gain access to the wound.

The relative incidence of rabies occurring in different animals is given by Stimson in tables collected from different sources. The dog leads with 80 per cent to 90 per cent of all cases, followed by the other domestic animals and finally the wild animals. At the Hygienic Laboratory of the United States Public Health Service over a ten year period, 588 animal heads were diagnosed positive for rabies of which 514 were dogs, 34 cats, 32 cattle, 5 hogs and one each, sheep, horse, and man. At the laboratory of Illinois State Department of Public Health over a four year period there were 416 positive animal heads of which 393 were dogs, 18 cats, 2 cows, 2 skunks and 1 wolf (table 27).

Table 28

COMPARATIVE REPORT ON 1494 ANIMAL HEADS RECEIVED IN OKLAHOMA STATE PUBLIC HEALTH LABORATORY
1925 and 1926

Animals	Total received	Number examined	Number Positive
Dogs	1204	994	340
Cats	168	135	29
Cows	53	47	11
Horses	2	2	1
Mules	4	4	2
Rats	2	0	0
Squirrels	21	13	1
Skunks	16	7	0
Hogs	7	5	2
Wolves	6	4	1
Guinea pigs	3	1	1
Chickens	1	0	0
Human brains	1	1	0
Sheep	1	1	0
Prairie dogs	1	0	0
Turkeys	1	0	0
Monkeys	1	1	0
Rabbits	2	2	1

The incubation period of rabies varies, depending upon several factors—the kind of animal afflicted, the kind of animal responsible for the injury, whether the wound is near the head or not and the amount of virus entering the wound. Reichel has given the incubation period of rabies in the domestic animals as follows:

Dogs, fourteen to sixty days
Cats, fourteen to sixty days
Cows, fourteen to eighty days
Horses, twenty-one to ninety days
Hogs, twenty-one to sixty days
Sheep, twenty-one to sixty days
Goats, twenty-one to sixty days
Birds, fourteen to sixty days
Rabbits, nine to ninety days
Guinea pigs, eight to sixty days

It is very rare for rabies to develop after the one hundredth day from the bite, although a few such instances have been reported.

An individual immunity seems to be present occasionally among dogs and other animals and large injections of virus do not affect them. A heriditary immunity of a transient character may be passed by an immune mother to her offspring. Natural immunity is enjoyed by only certain of the lower vertebrates as the reptiles. Under ordinary circumstances, death is the inevitable result when an animal becomes infected.

A few authentic cases are on record of recovery of dogs from an actual attack of the disease. They are very rare.

The dog is the natural reservoir of rabies, from which the disease spreads to other animals. The dog manifests symptoms in two different forms which are designated as *furious* rabies and *dumb* or *paralytic* rabies. The explanation for these differences is possibly because in the dumb type the toxemia is so overwhelming that the terminal stages of the disease are reached rapidly so that the furious symptoms do not manifest themselves.

Furious rabies in the dog is responsible for the spread of most cases of the disease. After a stage of development lasting one or two days, in which the animal may be unusually depressed or unusually affectionate, he has an irresistible tendency to roam. Many miles will be covered in the next two or three days, during which time he will bite anything that happens to come in his way. Rarely however, does he willfully attack man or other animals that are not in his immediate path. He will attempt to eat indigestible articles as sticks, stones, rags, etc. His appearance may not be abnormal enough to attract attention, though usually he looks like a sick animal. The foaming at the jaws, which sometimes appears, results from an accumulation of saliva due to difficulty in swallowing coupled with constant "champing" of the jaws. When the animal returns home from his roaming, he seeks a secluded spot and develops paralysis and soon dies.

Infrequently a dog presents no signs of frenzy or irritability and the first symptoms noticed are those of paralysis—so-called "dumb rabies." The animal hides away, develops paralysis in the lower jaw, followed quickly by the rest of the body and dies in one to three days.

Cats develop rabies with considerable frequency and with danger to human beings, especially children. The animal hides itself in a dark corner or under the furniture from which it sallies forth in vicious attacks on persons who come near. It will jump up and severely scratch the face of a child. The voice is lost, the animal mewing hoarsely. The appetite is later lost, with emaciation, paralysis and death in a few days.

Cattle are quite commonly affected with rabies and are capable of doing considerable damage due to their size and strength. Both the furious and the dumb types may occur, the former being the more common. After a preliminary stage marked by a change in the disposition of the animal, together with a loss of appetite, lessened flow of milk and restlessness, the furious type develops when the animal paws the earth, attacks everything in sight with its horns and bellows loudly with a peculiar change of voice. About the fourth day it becomes more quiet and develops paralysis, dying in a day or two more.

It is said that skunks may propogate the disease widely among their own kind. It is not uncommon to receive skunks in the laboratory for examination for rabies. Skunks have been known to attack persons also. According to Stimson animals other than dogs and skunks do not transmit the disease, as a rule, more than one remove from the dog. The relative inability, inopportunity or lack of tendency to bite when rabid is given as the cause for this.

Rats and other rodents are very susceptible, but it is unlikely that the disease is very often propagated among them. Their rather limited contact with man renders them of small danger.

Birds, such as fowls and pigeons, are susceptible to inoculation but show a relative immunity, many of the older ones recovering. Injection of brain material from such recovered birds will cause the disease in other birds but not in rabbits. The high body temperature of birds, 108° F., may be the cause of apparent immunity.

Cold blooded animals are not very susceptible. Several workers, among them von Lote and Marie, succeeded in infecting frogs but the incubation period was extremely long; negative results were obtained by other investigators. The tortoise is refractory to rabies.

The Disease in Man

Rabies in man follows a course not unlike that in animals. Both the dumb and furious types are met with. The incubation period may vary from fourteen to ninety days, according to the location and extent of the wounds. If the head and face are badly lacerated, symptoms may appear in as short a time as ten days; if the wound is on an extremity and slight, symptoms may be delayed for weeks or months. Several cases of an incubation period over a year have been reported. Women show a shorter incubation period than men.

Many persons bitten by rabid animals escape the disease, even without treatment. The virus may be wiped off from the teeth by the clothing or there may be an individual immunity among certain persons. At the Pasteur Institute in Paris it was found that only 16 per cent of persons bitten by rabid animals, but receiving no treatment, contracted the disease. Bites on the exposed surfaces, such as the face and hands, are much more dangerous than bites on other parts of the body. It is common experience that a bite on the hand is almost as dangerous, with an incubation period as short, as when the wound is located on the head.

The incidence of rabies in man is fairly well indicated by the number of deaths reported, since the disease is invariably fatal. For the ten year period 1915-24 in the United States Registration Area, human deaths have varied from 36 to 66 per annum.

During this same period investigations and surveys have been made by various individuals. Sellers for the period 1917-21 found 168 human deaths in 28 states or an average of 33 per year. Eichhorn's figures for 34 states in 1921-23 were 35, 34 and 37 per year. My own studies, as well as those of Rice and Beatty, indicate that human deaths are increasing in the United States. In 1926 Beatty reported 92 deaths.

Table 29

HUMAN DEATHS FROM RABIES IN THE UNITED STATES.

	U. S. Registration Area	Other Sources of Information
1915	52	
1916	36	
1917	66	Sellers, 1917–1921 28 States, total 168 Average per year of 33
1918	63	
1919	58	
1920	41	
1921	54	35 (Eichhorn 34 States)
1922	46	34 (Eichhorn 34 States)
1923	55	37 (Eichhorn 34 States)
1924	59	72 (Hull 36 states)
1925	—	111 (Hull 36 states)
1926	—	68 (Hull 36 states)

From the standpoint of mortality, rabies is a disease of childhood according to figures gathered from the United States Registration Area by the Metropolitan Life Insurance Co. Six deaths out of every ten from this disease occur among children under fifteen; more deaths occur between five and ten years than for any other age group. Boys are more often victims than girls; seven out of ten deaths of children under fifteen years are males. Not only are children more often attacked by rabid dogs than adults, but the incubation period in children is shorter, allowing less time for protective inoculation.

The control of rabies in man rests entirely upon prevention. After symptoms of the disease appear there is no known cure. Anti-serums have been tried by several investigators. Babes in 1890 used serum alone while Marie used an anti-sheep serum mixed with vaccine. Chicken serum has also been tried, but results have been unsatisfactory.

Prevention of Rabies

The prevention of rabies in man may be accomplished by either of two methods—the treatment of persons bitten by rabid animals or the elimination of rabies in dogs.

The Treatment of Persons Bitten by Rabid Dogs

The efficacy of anti-rabic vaccine has been demonstrated many times. At the Pasteur Institute in Paris from 1886 to 1905, 29,101 patients were treated with 122 deaths, a mortality of 0.41 per cent; from 1906 to 1924 16,741 patients were treated with

29 deaths—a mortality of 0.17 per cent; from 1925 to 1927 there were 2,055 patients treated with no deaths (table 25). At the Pasteur Institute in Chicago from 1890 to 1910 there were treated 4,463 patients with a mortality of 0.13 per cent. From 1910 to 1927 there were 2,467 patients with no deaths. The usual cause for death after treatment is delay in beginning the injections. Deaths occurring within fifteen days after treatment cannot necessarily be charged against the treatment. Other possible causes for failure are several. There may be a particularly massive dose of infection introduced into the wound or the strain of virus may be of unusual virulence. The response of the individual person to vaccine treatment must be likewise taken into account. While in rabies there are no statistics, in other diseases occasional persons are found who do not become immunized with a single series of injections. Immunity, as all other biological phenomena, is relative and not absolute.

The period of protection gained by a course of anti-rabic treatments is not very long. Experimental work with dogs would indicate that a course of treatments affords protection for eighteen months in that animal. In man the time is shorter. Park describes a case in which the immunity did not last fourteen months. The man, employed in a hospital for dogs, received an eighteen course treatment after a slight wound from a rabid dog. Thinking himself immune he became very careless, one day putting an abraded hand into the mouth of a rabid dog. Six weeks later he developed hydrophobia—fourteen months after his course of treatments. Sivori-Rebaudi has used the power of the blood serum to neutralize rabies virus as an indication of the immunity of an individual. Maggiulli found that in man the highest potency of the serum was reached thirty-five days after completion of treatment. Earlier than eighteen days after beginning of treatment or later than one year, the serum of vaccinated persons acts just the same as that of unvaccinated persons.

The unfortunate accompaniment of anti-rabic treatment is an occasional case of paralysis with sometimes fatal results. Various factors have been ascribed as the cause of this condition. Roux, Laveran and other early workers considered the symptoms due to an abortive type of rabies, modified from the usual symptoms by the course of injections. Fabricius and Nedrigailoff and Ostrijanin as well as others found that paralysis sometimes occurred when the wounds were slight or even when the biting animal was not rabid. Other explanations advanced are that it is a fixed-virus infection, that outside infection is introduced with the virus, that hysteria is responsible for the symptoms or that rabies "toxin" may cause the paralysis. Reasonable doubt exists that any of these are factors. Anaphylaxis, due to injection of foreign protein was suggested by Harvey and McKendrick, who called attention to the fact that the Högyes vaccine, prepared by dilution of the nerve substances, caused the least number of cases of paralysis. This view is confirmed by other workers who have found that injections of nerve substances from normal animals may cause paralysis. Simon (11) in 1913 collected figures in 92,950 cases which had received anti-rabic treatment. The original Pasteur method was used in 32,676 cases with 6 paralyses—1 in 5,446; the modified Pasteur method was used 8,657 times with 16 paralyses—1 in 541; the Högyes method was used 51,417 times with only 3 paralyses—1 in 17,139. Mejio in 1917 published figures on 19,800 cases treated at the Pasteur Institute, Buenos Aires with 24 paralyses and 4 deaths. At the New York City Health Department among 6,738 cases treated there were seven cases of paralysis and two deaths.

The following well-known rules for the care of suspected animals and recommendation for anti-rabic treatment are given:

> When a dog acts suspiciously, or when he bites a person, he should not be killed. On the contrary, the dog should be securely chained in a safe place and provided with his regular food supply during an observation period of two weeks. If available, a veterinarian should observe the dog. If the dog remains well and healthy throughout the two weeks, he may be released and any person whom he may have bitten need have no fear of coming down with rabies because of the bite. If, on the other hand, the dog should manifest the symptoms of rabies during the observation period, he should be killed so that the head may be detached without mutilation, packed in ice in a double tin lined container and expressed to the laboratory. After the results of the examination are known, plenty of time will remain for starting the Pasteur treatment in any humans who may have been infected. (An exception may be made of a bite of an exposed part without clothing. It is well known that the period of incubation is much shorter for bites on the face and hands. In such cases treatments should begin at the earliest possible time.)

How to Treat Dog Bites

First—Call a physician. The doctor will properly cleanse and dress the wound so that the possibility of infection from any disease, including rabies, will be less likely to occur than otherwise.

Second—The dog should be captured alive, if possible, and placed under observation after the manner prescribed above. In case the dog is killed, for any reason, his head should be detached without mutilation and forwarded to a laboratory where examinations for rabies can be made.

Third—If the dog is known to be rabid, the Pasteur anti-rabic treatment should be started at once. If rabies develops in the dog during the period when he is under observation, or if the diagnostic examination of the head shows the presence of rabies, the Pasteur treatment should be started at once upon learning either of these facts. In case rabies cannot be positively ruled out, even though it cannot be definitely established, the Pasteur treatment ought to be given as a precaution.

Who Should Take Preventive Treatment

The fact that a person is or has been bitten by a dog does not mean that he should take the Pasteur treatment. The Pasteur treatment is necessary and is recommended for only those persons who have been exposed to the bite of a rabid animal.

Persons who should take the Pasteur anti-rabic treatment may be classified as follows:

(a) Those who have been bitten, scratched or otherwise wounded by an animal KNOWN to be rabid.

(b) Those who have fresh open wounds in the skin which have been exposed to the saliva of an animal KNOWN to be rabid.

(c) Those who have been bitten or otherwise wounded by a sick animal that has exhibited the symptoms of rabies even though a definite diagnosis of rabies has not and cannot be made.

(d) Those who have been bitten by apparently healthy animals that were subsequently destroyed or for other reasons have not or cannot be observed in the manner described.

Cautery of wounds was a common practice before the discovery of anti-rabic vaccine. A red hot iron was the means of accomplishing the procedure. Fuming nitric acid has been found to be the only other efficient agent. Tincture of iodine comes next but is much less efficacious. Costill summarizes:

> Admitting the fact that cautery with fuming nitric acid will kill the virus of rabies, if—but only if—the acid comes directly in contact with the virus, there is still much to be said for the plea of those who argue that cautery should have no place in the treatment of bites of rabid animals. The reasons advanced against the practice of cautery are:
>
> (1) Only fuming nitric acid can be relied upon as an effective cauterizing agent and the pain caused by a thorough application of the acid may be great enough to require cautery under an anesthetic. Any cauterizing agent less painful cannot be relied upon.
>
> (2) In deep puncture-wounds it is impossible to know whether or not the cauterizing agent has reached the bottom of the wound and so killed the virus.
>
> (3) In extensive lacerations, cautery with fuming nitric acid is so destructive to the tissue that its use is not advisable.
>
> (4) Cautery may seal or wall in the virus of rabies; whereas unimpeded bleeding may help to wash the virus from the tissue.
>
> (5) Since cautery usually is ineffective, it gives a false sense of security and tends to prevent or delay the beginning of the Pasteur treatment.

The Elimination of Rabies in Dogs

This is a much more far reaching and efficacious method of preventing rabies in man.

The methods of elimination now used are two—restraint and vaccination. In either case it is necessary to destroy ownerless dogs. This is an expensive and unpleasant task, often involving the city authorities in controversies with irate but neglectful citizens who have lost their pets. In many cities where other preventive measures have been taken with great care, much of the effort is nullified because the important factor of the stray animal is neglected.

The restraint of dogs can be accomplished by locking up the animal, leashing it when on the street or muzzling it so that it cannot bite other animals. The confinement of dogs to the premises of the owner is a temporary measure for regions badly infected. It should last for at least three months, however, so as to cover the incubation period of any animals that might have been exposed, as well as to give the authorities opportunity to eliminate stray dogs. Leashing when on the street should be combined with muzzling since evasions are so numerous as to make muzzling necessary. Muzzling must be well supervised, to insure not only an efficient muzzle, but a humane one. The metal muzzzle of the basket type, properly fitted and changed from time to time with the animal's growth is very suitable. This factor is second only to the destruction of stray dogs in importance. It should be enforced over a sufficiently long period and wide area to insure absolute elimination of rabies before it is given up.

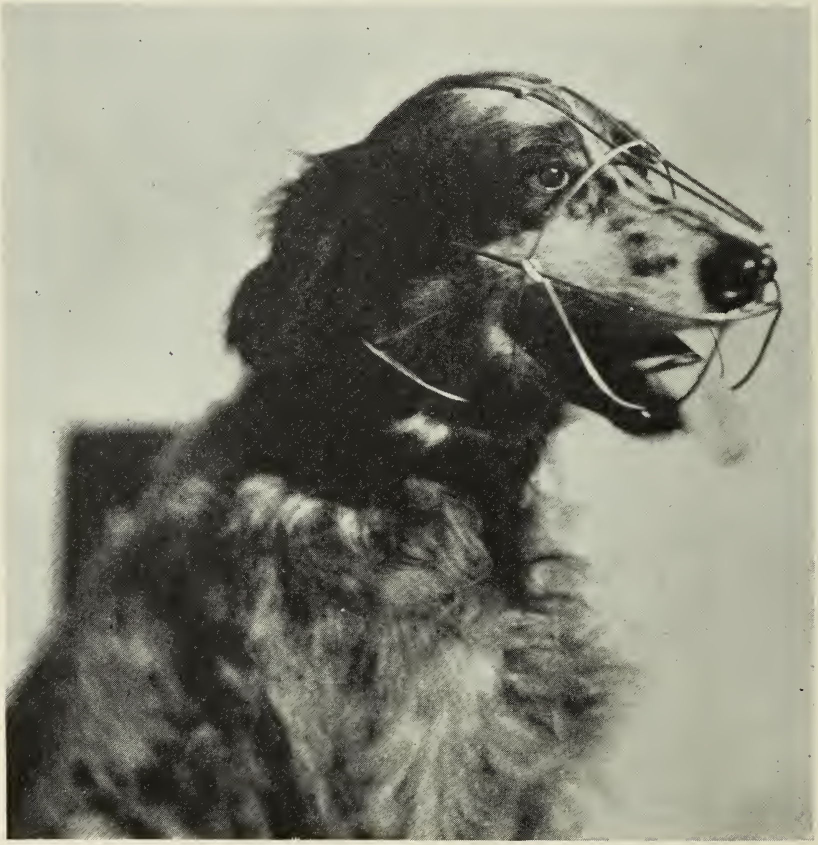


FIG. 17.—Proper type of dog muzzle, allowing dog to open its mouth for respiration while still preventing it from biting.

The efficacy of the control of rabies through restraint of the dog has been demonstrated in many communities. In England it has twice been used to free that island from infection.

Vaccination of dogs against rabies with one subcutaneous injection dates from the work of Umeno and Doi in Japan in 1921. They used a glycerinated fixed rabies virus attenuated with carbolic acid with very satisfactory results. Hata (12) in 1924 published the results of the first 104,629 dogs so treated in Japan.

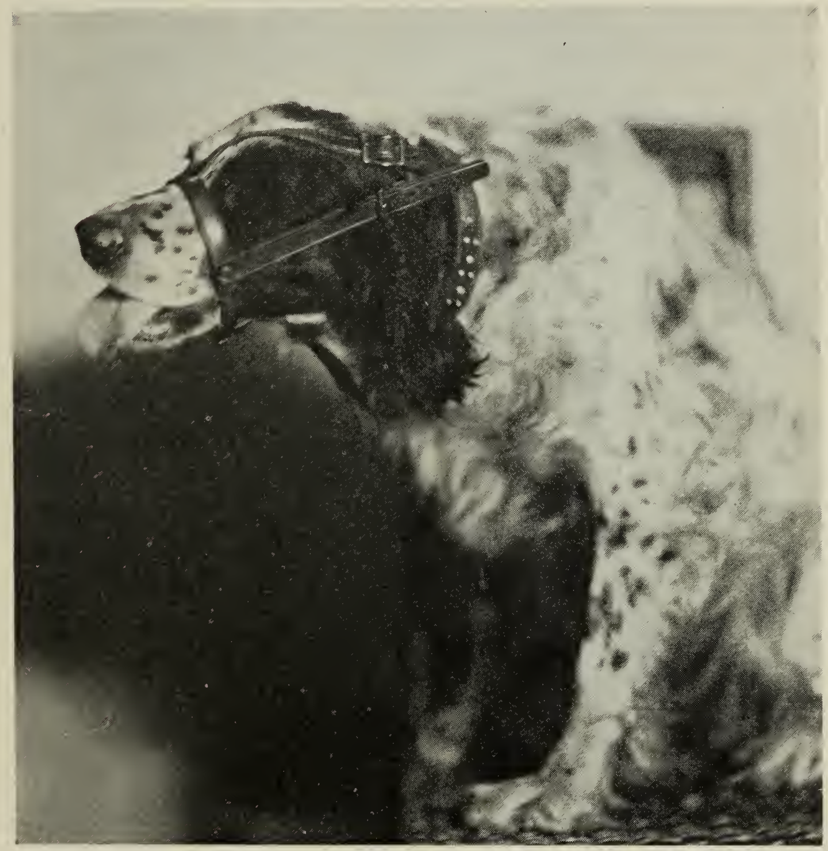


FIG. 18.—Improper type of dog muzzle, which only serves to irritate the dog and in no way prevents it from biting.

Only 41 of the inoculated animals developed rabies, most of which had been exposed before vaccination was undertaken. In the control group of less than one-third the vaccinated group, 1,699 dogs developed the disease.

Experimental work by investigators in the United States has established the value of the Japanese method. Eichhorn and Lyon

(13) immunized six dogs with single injections of 5 c.c. of vaccine. Three weeks later these dogs were inoculated intra-ocularly with a suspension of brain tissue from three different dogs known to have died from street rabies. At the same time three control dogs, unprotected by vaccine were similarly infected with street virus. The six vaccinated dogs did not develop rabies while the three controls died of the disease in fifteen to seventeen days. The work of Reichel and Schneider and of Schlingman was equally conclusive. In Austria Schnurer and in Uruguay Schern have also had good results.

Schoening at the Bureau of Animal Industry of the United States Department of Agriculture showed that one injection of vaccine afforded distinct protection against rabies under severe laboratory conditions. Several lots of street virus from different sources were used to infect both the vaccinated and unvaccinated dogs through the anterior chamber of the eye. In one experiment with a virus designated B.A.I. 474, vaccine failed to protect, indicating that possibly there is in this country more than one strain of street virus. A polyvalent anti-rabic vaccine may eventually be necessary. Experience with human anti-rabic vaccination would not indicate this however. Practically all laboratories making anti-rabic vaccine use the same strain of fixed virus, yet failures are almost negligible in man.

Other failures in dogs with the single injection of anti-rabic vaccine have been noted from time. In a survey which I made in 1927 a few cases were reported (2). In Ohio seven dogs developed the disease after vaccination, five of which showed symptoms within two days and the other two after three to six months; Arizona, one dog after six months; Tennessee, three dogs after two to three months; New Jersey, one dog after nine months; Virginia, two dogs after four to eight months; Washington State, one cat after one year; New York State, two dogs after three to six weeks, two dogs after four to six months and one dog after a year. In Georgia, where in one year about 7,000 dogs were vaccinated, some cases of failure had been reported but no histories had been obtained.

An attempt was made to compare the effect of vaccination of dogs on the yearly demand for human anti-rabic treatment. The annual report of the Georgia State Board of Health for 1925 states: "In the first six counties there appears to be a possible decrease in 1925 in rabies as indicated by the decreased demand for treatment. In the last four counties listed rabies remained stationary or increased in 1925. Hence no definite conclusion can be drawn."

Compulsory anti-rabic treatment of dogs has been gaining in favor in many places. In Japan it became effective in 1923. In Uruguay such a bill was introduced in 1926. In the United States in 1927 in fifty cities in thirteen states vaccination was compulsory, while several counties had similar laws. The State of Washington required all dogs shipped into the State to be vaccinated, with the exception of performing dogs. In New Mexico the State Director of Public Health could order all dogs within any area specified by him to be effectively muzzled, or confined, or else be impounded or killed, unless protected by inoculation with a vaccine of a recognized potency. In a large number of other communities anti-rabic vaccination of dogs had been recommended by health authorities and veterinarians and in many instances it had been made optional to other methods of control, as in New Mexico. Eichhorn estimated that between 1923 and 1926 half a million dogs had been vaccinated in the United States (16), and by 1928 at least two million dogs (19).

In Illinois I studied histories of 454 rabid dogs in 1928, finding 18 which had previously been vaccinated. The intervals between vaccination and the first symptoms of the disease follow:

2 dogs	1 day interval	1 dog	5 weeks interval
1 dog	4 days interval	2 dogs	6 weeks interval
2 dogs	2 weeks interval	1 dog	2 months interval
2 dogs	3 weeks interval	1 dog	3½ months interval
1 dog	4 weeks interval	1 dog	4 months interval
2 dogs	1 month interval	1 dog	6 months interval
1 dog	time not given		

It is apparent that the animals developing the disease within a few days after vaccination were in the incubation stage when vaccinated and one injection afforded insufficient protection. Whether the animals which developed the disease three to six months later were in the incubation stage when vaccinated is impossible to state. The inference is that these constitute cases where the vaccine failed, but it may not be so. The vaccination may have had some effect upon lengthening the incubation period.

The extent to which anti-rabic vaccination has been a factor in the control of rabies is difficult to state because careful observations in most instances have not been carried out.

The rôle of the stray dog in the spread of rabies is indicated in a study which I made in Illinois. During the summer of 1927, information was collected upon 115 dogs which had shown positive laboratory examination at various laboratories in the state. Of the 50 stray dogs, only 5 had been under the observation of a veterinarian, while 35 of the 65 whose owners were known had been so observed. It would appear that the stray dog was a serious menace in spreading rabies among other dogs for the 50 stray dogs were known to have bitten 108 other dogs (and prob-

Table 30

INFORMATION ON 115 DOGS WHICH SHOWED POSITIVE LABORATORY EXAMINATION FOR RABIES

Laboratories	Stray Dogs	Owners Known
Springfield	18	29
Carbondale	4	12
Urbana	1	1
Chicago	27	23
Total	50	65

UNDER OBSERVATION OF VETERINARIAN

Laboratories	Stray Dogs	Owners Known
Springfield	3	18
Carbondale	1	
Urbana		1
Chicago	1	16
Total	5	35

PERSONS AND ANIMALS BITTEN BY RABID DOGS

Laboratories	Bitten by Stray Dogs			Bitten by dogs whose owners were known		
	Persons	Other dogs	Other animals	Persons	Other dogs	Other animals
Springfield	13	75	6	21	36	9
Carbondale	5	19	1	19	21	
Urbana						
Chicago	27	14	1	40	5	3
Totals	45	108	8	80	62	12

ably more), while the 65 home dogs bit only 62 others (and probably but few others for many of them were under the observation of a veterinarian). The home dog is more of a menace to persons however, for they bite nearly twice as many people as do stray dogs. It would appear therefore, that for the protection of the family, anti-rabic vaccination of the home dog is a wise precaution.

The Problem of Milk and Meat

Rabies virus has been found in the milk of infected animals by Nocard, Perroncito and Bardach. It probably is present in the milk several days before the animal shows symptoms, as it is in the saliva. Mohler inoculated the milk of a rabid bitch into guinea pigs, producing typical symptoms. The puppies of the bitch removed from her at sign of the first symptoms, remained well and healthy. It is generally conceded that there is no danger from the ingestion of infected milk unless there be abrasions on the lips or along the alimentary tract through which the virus may enter the circulation. Mohler cites the case of a woman in the early stages of rabies who transmitted the disease to her nursing baby apparently through the milk, but the possibility of abrasions of the gum through teething must be considered. Other instances are on record, where nursing animals have become infected apparently through the milk, but the possibility of abraded gums from teething or scratches and bites of the mother cannot be ruled out. Milk from cattle suspected of rabies should not be used for human consumption or fed to animals. Cows in herds that have been attacked by rabid dogs, but where infection is doubtful, should be carefully watched for possible symptoms, the milk being boiled or pasteurized meanwhile.

Persons with cuts on their hands should not milk such cattle as the incubation period draws to a close.

Meat from rabid animals as well as brain material has been fed to dogs with no ill results. Wyrsykowski showed that the gastric juice quickly destroys the virus. He digested brain material with gastric juice and injected twenty-one rabbits, none of which developed rabies; seventeen check rabbits injected with undigested material, all developed the disease.

The use of meat from cattle infected with rabies would be unwise, and probably would not take place if the symptoms were well developed. It seems to be the custom in some localities, when rabies appears in a herd of cattle, to rush those animals not already showing symptoms to the slaughter house to be converted into meat. Since the saliva is infective several days before symptoms develop it is probable other portions of the body must be infective. Butchers and others handling such meat would be placed in danger of infection if abrasions were present on the hands. Proper cooking would destroy the virus.

Items of Note

1. Rabies is a disease to which all warm blooded animals, without exception, are susceptible.
2. The dog is responsible for the spread of rabies to other animals and to man.
3. Elimination of rabies in dogs serves to eradicate the infection in that vicinity.
4. The virus of rabies is probably protozoan and not bacterial in nature.
5. After symptoms of rabies appear there is no cure.
6. Not more than one person in six bitten by rabid animals develop the disease, even though no anti-rabic treatment is taken.
7. Bites on the exposed surfaces, as the face and hands, are most dangerous.
8. Rabies is an abrasion infection requiring a broken skin for entrance of the virus.
9. Ingestion of the virus is probably not dangerous unless lesions are present in the mouth or throat. The gastric juice of the stomach quickly destroys the virus.
10. Milk from cows suspected of having been exposed to rabies should be thoroughly pasteruized or boiled.
11. Rabies is an all the year around disease, occurring during every month of the year.
12. Anti-rabic vaccine can be considered a sure preventive against the disease in man when administered promptly. It should not be administered to persons who do not need it.
13. The elimination of rabies in a community requires the restraining or muzzling of all dogs accompanied by a strenuous campaign to destroy all stray and ownerless dogs. Other methods of control can be used to supplement, but not to replace these measures.

BIBLIOGRAPHY

1. Stimson, A. M.
   U. S. Public Health Service, Hygienic Laboratory Bull. 65, June, 1910.
2. Hull, T. G.
   Nations Health, June, 1927, *9*, 21-24.
3. Rice, T. B., and Beatty, N.
   Am. Jour. Public Health, 1928, *18*, 421-428.
4. Williams, A. W., and Lowden
   Jour. Inf. Dis., 1906, *3*, 452.
5. Calkins, G. N.
   Proc. N.Y. Path. Soc., 1906.
6. Noguchi, H.
   Jour. Exp. Med., 1913, *18*, 314, 678.
7. Kelser, R. A.
   Jour. Am. Vet. Med. Asso., 1924, *64*.
8. Levaditi, C., Nicolau, S., and Schoen, R.
   Ann. de l' Inst. Past., 1926, *40*, 573.
9. Schoening, H. W.
   Jour. Agr. Res., 1925, *30*, 431.
10. Park, W. H., and Williams, A. W.
    Pathogenic Microörganisms, 8th Edition. Lea and Febiger, Philadelphia.
11. Simon, G.
    Centralbl. f. Bakt., 1913, Orig. *68*, 72.
12. Hata, S.
    Jour. Imm., 1924, *9*, 89.
13. Eichhorn, A., and Lyon, B. M.
    Jour. Am. Vet. Med. Asso., 1922, *61*, 38.
14. Reichel, John
    Jour. Am. Vet. Med. Asso., 1923, *63*, 83.
15. Schlingman, A. D.
    Jour. Am. Vet. Med. Asso., 1925, *68*, 299-305.
16. Eichhorn, A.
    Am. Jour. Public Health, 1926, *16*, 644-647.
17. Schoening, H. W.
    Jour. Agri. Res., 1925, *30*, 5.
18. Viala, J.
    Ann. de l' Inst. Past., 1928, *42*, 724.
19. Eichhorn, A.
    Jour. Am. Vet. Med. Asso., 1928, *72*, 1023-1029.

CHAPTER X

PSITTACOSIS

Psittacosis is primarily an infectious disease of parrots and parrakeets characterized by fever, stupor, debility and diarrhea. Secondarily man may become infected, the disease developing into a severe pneumonia.

HISTORY

Psittacosis as a disease of parrots has been known for a long time. It attracted no attention however until 1879, when Ritter (1) traced a small epidemic of pneumonia in several families in Switzerland to infected parrots imported from Germany. Similar instances were reported by Ost and by Wagner in 1882 and again in 1886. In 1892 an epidemic of severe pneumonia occurred in Paris which was attributed to diseased parrakeets imported from Buenos Ayres. Nocard, who investigated this epidemic, isolated from human cases as well as from sick birds an organism supposed to be the causative agent, which he named *Bacillus psittacosis* (2). Since that time Nocard's findings have been confirmed on various occasions. Palamidessa in 1895 isolated the organisms from five human cases in one family in Florence. Gilbert and Fournier in 1896 isolated it from the blood of an infected woman as well as from parrots. Other cases in France, England (3), Germany (4), and America have likewise been reported (5).

THE CAUSATIVE AGENT

Salmonella psittacosis (*Bacterium psittacosis* of Nocard) is a small rod 0.4 by 0.6 microns wide and 1.0 to 1.5 microns long. It grows upon ordinary culture media, is motile and Gram negative. It belongs to the Salmonella or paratyphoid group of organisms (7).

There has been some doubt as to whether this organism was truly the cause of psittacosis or not. Most observers have failed to find it in human cases, finding rather pneumococci, streptococci, colon bacilli, or other organisms. Nocard believed that Salmonella psittacosis paved the way for the other organisms to gain a foothold. The work of Bachem, Selter, and Finkler (6) pointed to a streptococcus as the causative agent. Gulland (3) found pneumococci in an epidemic among parrots in England.

The Disease in Parrots

Psittacosis in parrots is a highly fatal infection, from 50 per cent to 95 per cent of infected birds dying. Parrots are very susceptible, healthy birds being placed in infected cages or in clean cages which contain no more than a few infected feathers, invariably contracting the disease. Contaminated food, water, or other utensils may also cause the disease, as well as the soiled hands of an attendant. Mice, or other rodents or insects, may mechanically carry infection on their feet from one cage to another.

Infected birds show a general debility, loss of appetite, and chronic diarrhea. They rapidly become emaciated and light, dying in a few days. Salmonella psittacosis is found in all organs, the blood, and bone marrow of dead birds. The liver, spleen, and kidneys show cloudy swelling, but there usually is no involvement of the lungs except in birds which live for some time before dying.

Besides the parrot and parrakeet, other birds and animals may be infected by artificial inoculation, as the fowl, rabbit, mouse, and guinea pig.

The Disease in Man

Gulland (3) draws attention to the striking fact that a disease, which is obviously of the abdominal type in the bird, is seldom associated with abdominal symptoms in the human being, but takes the form of an atypical pneumonia.

The disease is mostly confined to those persons who come into contact with sick parrots. Man has quite a high susceptibility to the disease, as evidenced by the fact that it is quite common to find all members of a family sick, wherever the disease has gained entrance. The mortality in such house epidemics is rather high. In the Paris epidemic of 1892 there were 49 cases with 16 deaths. From 1892 to 1897 Dupuy found that 70 cases of the disease occurred in Paris with 24 deaths, a mortality of 34 per cent.

Prevention

Prevention of psittacosis depends upon sanitary precautions by those persons who are liable to come into contact with sick parrots.

The disease has not been found to occur in the general population except as sick parrots were implicated.

Items of Note

1. Psittacosis is primarily a disease of parrots.
2. Man is susceptible to the disease, exhibiting the symptoms of an atypical pneumonia with a high mortality.
3. The disease is confined largely to those persons who come into contact with sick parrots.
4. When a member of a family has become infected, the disease may be passed to the rest of the household, indicating a high susceptibilty of man.
5. Prevention rests upon sanitary precautions by those persons liable to come into contact with sick parrots.

BIBLIOGRAPHY

1. Ritter, F.
Deutsch. Arch. f. Klin, Med., 1879, *25,* 53.
2. Nocard, E.
Conseil d. Hygiene Publique et Salubrite du Dept. du Seine, Seance, March 24, 1893.
3. Gulland, G. L.
Brit. Med. Jour., 1924, *2,* 309.
4. Leichtenstern
Centralbl. f. allg. Gesundh., 1899, *18,* 241.
5. Vickery, H. F.
Med. News, 1904, *85,* 780.
6. Bachem, Selter, and Finkler
Klin. Jahrb., 1910, *23,* 539.
7. Perry, A. M.
Brit. Jour. Exp. Path., 1920, *1,* 131.

CHAPTER XI

FOOD POISONING

Certain members of the Salmonella group of bacteria are associated with symptoms of food poisoning characterized by nausea, vomiting, abdominal pains, and diarrhea. Several of these organisms are of animal origin, being transmitted to man by way of the food supply.

The term "food poisoning" is unsatisfactory in many ways; the term is indefinite in meaning, having been used to cover a variety of derangements which were not due to the food at all but rather to functional disorders; the term is inclusive in meaning, having been used to cover a variety of derangements due to poisonous substances transmitted by way of the food but not of bacterial origin. The term "food poisoning" is infinitely better than the term "ptomain poisoning" which is entirely unsatisfactory and has no basis for existence.

Several forms of disturbances are sometimes classified as food poisoning which should not be so included.

(a) Disturbances due to individual hypersensitiveness to certain foods, such as milk, eggs, strawberries, tomatoes, oatmeal, etc.

(b) Disturbances caused by poisonous plants or animals, such as toadstools, water hemlock, or poisonous fish.

(c) Disturbances caused by chemicals which have found their way into the food by accident or intention, such as arsenic, antimony, lead, tin, or copper.

(d) Communicable diseases spread by way of the food supply, either from a carrier or case, such as diphtheria, typhoid fever, scarlet fever, etc.

(e) "Ptomain poisoning" so-called, supposed to be due to decomposed and putrid food, but which probably does not exist. It is doubtful whether putrid food, in the absence of specific germs, will cause food poisoning.

Food poisoning, in its technical sense, is confined to certain infections and intoxications associated with specific bacteria.

(1) Food infections and intoxications caused by members of the Salmonella group of bacteria (Gaertner group), which are characterized within a few hours after the ingestion of the offend-

ing food by symptoms of nausea, vomiting, abdominal pains, and diarrhea.

(2) Intoxication caused by the toxin of the botulinus bacillus, attended by an attack upon the central nervous system but without an acute gastro-intestinal upset.

Botulism is differentiated from other forms of food poisoning in that (a) it is caused by an otherwise harmless saprophyte, (b) the organisms always gain access to the food by accidental contamination, (c) the toxin attacks the central nervous system instead of the gastro-intestinal system. Botulism is discussed in detail in Chapter 24, while the present chapter is confined to the Salmonella group.

History

Food poisoning is as old as civilization itself. From his earliest creation, down through the ages, man apparently acquired considerable knowledge concerning what was good to eat and what to let alone. By the fourth century, B.C., it was recognized that the bodies of animals dying a natural death were not fit for food, for Moses commanded that "Ye shall not eat anything that dieth of itself." Moses was not particularly concerned about the ethics of the matter, however, for he permitted that "Thou mayest give it unto the sojourner within thy gates that he may eat it; or thou mayest sell it unto a foreigner" (2).

Food poisoning mentioned by Hipprocrates, Horace, Ovid, and other ancient writers was often of a different nature, usually having to do with poisonous mushrooms or other poisonous foods. Later conceptions of food poisoning were purely chemical in nature. With the advent of bacteriology the term "ptomain" came into common use to cover all forms of food poisoning. The wide newspaper publicity of every case of botulism that occurred placed an undue emphasis upon this form of food poisoning in the minds of many persons, so that the term "botulism" and "food poisoning" were synonymous whatever the cause.

Prevalence

Accurate figures concerning the prevalence of food poisoning in the United States are lacking; compulsory reports of such instances are not required; many of the reports taken from the daily press are wholly unfounded and few of the actual outbreaks are carefully studied.

From 1910 to 1922 there occurred in the United States, according to Geiger (1), 749 outbreaks of food poisoning which were studied epidemiologically. During the next three years there were reported 425 more (2). There was a steady increase each year over the entire period. In 1910 there were reported to the United States Census Bureau 157 deaths from food poisoning, while in 1923 there were 928 such deaths reported, and a total for the fourteen years of 9,981 deaths. Many of these were probably due to other factors, however.

In the study of 1,174 outbreaks from 1910 to 1925 inclusive, there were 7,112 persons involved with 544 deaths, a per-centage mortality of approximately 7.6. There were included, however, six outbreaks of botulism in which 253 persons were made ill and 197 died. Deducting these deaths gives a mortality of 5.0 instead of 7.6. There were likewise 87 deaths from "ptomain poisoning" the diagnosis of which was changed after necropsy; there were 46 deaths from metallic poisoning, and 8 deaths from acute alcoholism. The deduction of these 141 deaths reduces still further the mortality percentage from 5.0 to 2.9. A careful study of the remaining epidemics was made in which only those were included where the laboratory studies or epidemiological evidence was sufficiently conclusive to consider them caused by organisms of the paratyphoid group. There were 120 such outbreaks, involving 3,299 persons with a percentage case mortality of 0.4. The rate of 2.9 can be further reduced to 0.4 per cent which approximates the mortality rate in most instances.

In England food poisoning is supposed to be more common than in the United States, and on the Continent of Europe still more prevalent. Savage tabulated 112 British outbreaks occurring between 1878 and 1918, while Savage and White (3) added 100 more, mostly between 1921 and 1923. In Germany Mayer reported 48 food poisoning outbreaks between 1888 and 1911, due to *B. enteriditis,* while Bainbridge found 261 outbreaks from 1898 to 1908 due to *B. enteriditis* and *B. suipestifer.* Klimmeck reported 43 outbreaks in Prussia in 1924, 64 in 1925, and 69 in 1926. In the 69 outbreaks there were 2,281 cases with 14 deaths.

The Salmonella Group

Much confusion in nomenclature is evident in the literature concerning the organisms which cause food infections. They have been variously known as the paratyphoid group, the Gaertner group, the Salmonella group, the hog-cholera group, the enteriditis group, and the intermediate group.

Bergey (4) lists 19 members of the Salmonella group, some of which may be identical. They are small gram-negative rods which are easily cultivated on the ordinary culture media. They are aerobes or facultative anaerobes. They all attack numerous carbohydrates with the formation of both acid and gas. They generally occur in the intestinal canal of man or animals in various types of acute inflammatory conditions.

Organisms causing food infection which are of animal origin follow:

Salmonella suipestifer
(*Bacterium suipestifer, Bacillus cholerae suum* associated with hog cholera, but not the cause of the disease.)

Salmonella enteriditis
(*Bacillus enteriditis* Gaertner. Widely distributed, occurring in both domestic and wild animals.)

Salmonella aertrycke
(*Bacillus aertrycke.* Closely related to *S. schottmülleri* and *S. suipestifer.* Encountered in continued fevers of the paratyphoid type.)

Bacillus pestis-caviae is a member of this group, considered by some to be identical with *S. aertrycke* but probably a distinct organism found usually associated with rodents.

Organisms of animal origin which possibly give rise to food poisoning are *S. gallinarum (B. sanguinarium),* the cause of fowl typhoid; *S. pullorum,* the cause of white-diarrhea of chickens; *S. abortus-equi,* the cause of contagious abortion of horses; and *S. anatum,* the cause of "keel" in ducklings.

Salmonella psittacosis, causing a severe intestinal upset in parrots, is transmitted to man by contact where it gives rise to an atypical pneumonia (see chapter 10).

Two organisms of the Salmonella group which cause food poisoning are of human origin. They are *S. paratyphi* (paratyphoid A) and *S. schottmülleri* (paratyphoid B). They are closely allied to the typhoid organism, are easily differentiated from the animal paratyphoids, and are found solely in the human intestine and never in lower animals or elsewhere. They will not be considered in this discussion therefore.

The remaining members of the Salmonella group are interesting as far as their relation to animal pathology is concerned, but have no relation to food poisoning and hence may be neglected.

A miscellaneous number of other organisms have from time to time been associated with outbreaks of food poisoning, but their etiological significance is not clear. Members of the proteus group are commonly found in decomposing food and several investigators have attributed food poisoning outbreaks to them. Savage, however, after studying nine such reported instances, concluded that "for none of them was it established that *B. proteus* was etiologically concerned."

Staphylococci and streptococci have likewise been incriminated. The evidence is not clear whether such organisms are capable of producing food poisoning. Occasional cases may also be caused by other bacteria, knowledge of which we are in ignorance at the present time.

Suipestifer Infection. Salmonella suipestifer (*Bact. suipestifer,* Bacillus of hog cholera, *Bacillus cholerae suum*) was isolated by Salmon and Smith in 1885 from pigs suffering from cholera and was thought to be the causative organism. It was the first organism of this group to be described, hence, by priority, the term Salmonella. It has been shown that the suipestifer organism is a secondary invader in hog cholera, the true cause of the disease being a filterable virus. It is an ordinary inhabitant of the intestinal tracts of hogs, to which no significance is attached under normal conditions. When the resistance of the animal is lowered it takes on special pathological significance as a secondary invader. Neither the cow nor the rat harbor it, although it has been isolated from the mesenteric gland of a monkey.

Infected pig meat is the usual source of epidemics of food poisoning although the channels by which the infection travels are obscure. The instances when man has been infected by the suipestifer organism are comparatively rare. Savage and White (3) encountered it only once in their 100 epidemics (see table 31). A still further remarkable fact is the absence of such infection in man on farms where hog cholera is common. Even consumption of meat from a hog infected with the disease does not necessarily produce illness.

Enteriditis Infection. Salmonella enteriditis (*Bacillus enteriditis,* Gaertner bacillus) was discovered in 1888 by Gaertner in Saxony, following the illness of 57 persons who had eaten the flesh of a cow that had been killed because it was sick. One man who consumed nearly two pounds of raw flesh died in thirty-five hours. From the organs of the man, as well as from meat he had eaten, Gaertner isolated the organisms causing the trouble. Other outbreaks have been reported from time to time in which similar organisms have been found carried by beef, veal, and milk. Savage and White (3) isolated it five times from thirty-one epidemics carefully studied in the series of one hundred epidemics —twice from porcine sources and three times from bovine. McWeeney (6) encountered it in beef stew.

Rodents are a common source of enteriditis infection. Spontaneous outbreaks among rats and mice, both under natural and experimental conditions, have been noted. The animals infect food by their droppings, causing severe outbreaks of food poisoning. Meyer in California (8) found 28 wild rats out of 775 examined harboring this organism. Verder (26) isolated it from the liver and spleen of four rats and the spleen of a fifth among 114 wild rats caught in Chicago.

Savage and Read (27) in England found 5 rats infected out of 41 examined. Savage and White (28) isolated 6 virulent strains from 96 rats caught at two slaughter houses where bacterial bait had not been used in over a year. They also found that the blood of 30 per cent of the rats gave positive agglutination reactions with enteriditis but not with aertrycke or suipestifer.

Table 31

ISOLATION OF SALMONELLA ORGANISMS IN VARIOUS EPIDEMICS OF FOOD POISONING

(Savage)

Type of Organism	Number times isolated	Food Vehicle				Unspecified or other vehicle
		Pig	Bovine	Sheep	Not meat	
Aertrycke	22	1	7	1	8	5
Newport (aertrycke)	3	—	1	—	1	1
Enteriditis	3	—	3	—	—	—
Derby (enteriditis)	2	2	—	—	—	—
Suipestifer	1	—	—	—	1	—

Commercial rat viruses usually contain the enteriditis organism; several human epidemics are traceable to such products (9).

Spray reports an outbreak involving 123 cases due to contamination of milk with a commercial rat virus (7).

Aertrycke Infection. Salmonella aertrycke (Bacillus aertrycke) is the most common cause of food poisoning. The organism was first isolated by DeNobele in 1889 from a food poisoning outbreak. It is closely related to *S. suipestifer,* with which organism the English bacteriologist long considered it identical. The German bacteriologists, on the other hand, confused it with *S. schottmülleri* (paratyphoid B) with which it was considered identical. Jordan (10) has clearly emphasized the distinguishing characteristics of *aertrycke* and *schottmülleri.*

Schütze (12) has divided the aertrycke group into seven different strains by means of absorption tests. The "Mutton" type is the largest, being encountered 24 times out of a total of 46 for the group. The others are Hirschfield type, Newport type, Stanley type, Binus type, Arkansas type (hog cholera), and G type. The literature is very much confused, however, requiring a careful analysis of each reported outbreak of food poisoning attributed to this cause.

The organism occurs occasionally as an inhabitant of the intestinal tract of cattle but not as often as does enteriditis. It reaches man through the meat of ailing animals. Sheep especially have been the cause of extensive epidemics. Swine do not seem to be implicated with this organism. Rats, mice, guinea pigs, and rabbits are not infrequently infected and may be a source of danger to man. Verder found it in the spleen of one rat out of 114 examined. Jordan (11) does not agree with Krumwiede that it is a distinct rodent type.

Numerous outbreaks of food poisoning have been attributed to *S. aertrycke.* Savage and White identified the organism 25 times in 31 outbreaks where the causative agent was isolated (table 31).

Other animal paratyphoids. Salmonella pestis-caviae is considered by some investigators to be identical with *S. aertrycke* (8). Others believe that it is a separate type, however. Salthe and Krumwiede (13) reported an epidemic involving 59 cases, due to this organism carried by corn starch cream filling which had been infected by rat droppings. They studied the organism isolated by Winslow and his co-workers (14) from egg salad, classifying it as *pestis caviae.* The organism is apparently a common inhabitant of the intestinal tract of rodents.

Salmonella abortus-equi is the cause of contagious abortion in mares. While several epidemics of food poisoning following the consumption of horse meat have been reported from Europe, there is no bacteriological evidence to incriminate this organism.

Salmonella pullorum was described by Rettger in 1908 as the cause of white diarrhea in chickens. The disease is very prevalent in poultry with over 25 per cent of recovered fowls as permanent carriers. The organisms are found in the eggs where they multiply to enormous numbers under the usual method of market distribution. The relation of the affection to man is not known, but several experimental demonstrations have been carried out which would indicate the possibility of a serious menace to man. Working in Rettger's laboratory, I found that young rabbits and kittens easily succumb to the infection (15).

Salmonella gallinarum (Bacillus sanguinarum) is the cause of "fowl typhoid" and is infectious for numerous small animals under experimental conditions. Its relation to man, however, is undetermined.

Kind of Food Responsible

Most outbreaks of food poisoning are due to some form of prepared meat, such as sausage, meat pies, chopped meat, and the like. Savage and White tabulated 112 outbreaks of bacterial origin occurring in Great Britain from 1898 to 1918, in only 21 of which the vehicle was a non-flesh food—milk one, cream

Table 32

VEHICLES OF FOOD POISONING IN 100 BRITISH OUTBREAKS
1923-25

Tinned foods (tongue 3, beef 15, mutton 1, salmon 15, sardines 2, crustacea 3, fruit 3)	42
Milk and milk products (milk 5, cheese 8, ice cream 1)	14
Made-up meat foods (brawn 2, potted meat 6, meat pies 3, sausages 2, rissoles 1, faggots 1)	15
Manipulated but not made-up meat foods (chicken broth, pickled beef, frozen oxtail, stuffed mutton, frozen beef)	5
Fresh meat	6
Fresh fruit and vegetables (tomatoes, plums, potatoes, apples)	4
Preserved fruit	2
Other food (cockles, linseed tea, shrimps, fried fish)	4
Multiple or not ascertained	8
	100

one, ice cream six, potatoes two, canned peaches one, pineapple jelly one, rice one. The number of cases ascribed to fish were small and to sheep almost lacking. Meat from the hog or the cow were responsible for 68 per cent of the outbreaks.

In Savage and White's 100 outbreaks occurring mostly between 1923 and 1925, commercially canned foods were responsible in 42 cases while manipulated meat foods were the cause of 20 others, and milk or milk products 14 (table 32).

In the 1174 alleged outbreaks occurring in the United States from 1910 to 1925, vegetables and fruits were reported as the responsible vehicle 591 times, or in more than half the cases. It must be remembered, however, that many of the instances included in that study were due to causes other than the Salmonella group of organisms (table 33).

Table 33

VEHICLES OF FOOD POISONING IN 1174 ALLEGED OUTBREAKS IN THE UNITED STATES
1910 to 1925

Vehicle	Food Commercially Preserved	Food Home Prepared	Total
Meat	68	80	148
Fish	324	6	330
Vegetables	413	67	480
Fruit	100	11	111
Soup	63	0	63
Milk	37	35	72

Many authentic records have been published indicating that a variety of foods may act as vehicles for food poisoning. Winslow reported egg salad; Salthe and Krumwiede corn-starch cream filling of éclairs; Stewart and Litterer, as well as Spray, milk; Rosenau and Weiss, bread pudding; Krumwiede, Provost, and Cooper, bread pudding. I encountered an outbreak where 24 cases were involved resulting in 3 deaths, due to the corn-starch cream filling of cream puffs.

Members of the Salmonella group grow rapidly in most foodstuffs without producing much evidence of their existence. Koser (16) found that all members of the group multiplied rapidly in the liquor of several cooked vegetables, with the exception of highly acid sauerkraut. In fruit juices a rapid destruction of the organisms took place. In several meat products the organisms exhibited a marked ability to spread from the original point of inoculation throughout the foodstuff, although this occurred only under optimum conditions. Damon and Leiter (17) had a similar experience, finding that under ordinary household conditions, the organisms will multiply to a considerable extent in 24 to 72 hours, producing such slight changes in color and odor as to be readily overlooked.

Animals Responsible for Food Poisoning

Cattle suffer from puerperal fever, diarrhea, septicemia, uterine inflammations, and the like, caused by members of the Salmonella group. In Europe it is customary to slaughter such sick animals for food, hence the high proportion of epidemics reported there due to meat. While Uhlenhuth has reported the occurrence of such organisms in the intestines of healthy calves and cattle, Jordan's (9) study on the intestinal contents of 52 normal animals killed at the Union Stock Yards in Chicago showed no organisms that fulfilled all the requirements of the food poisoning group. Friesleben (24) examined the intestinal tracts of 100 oxen, 100 calves, 50 sheep, and 100 pigs. Organisms belonging to the paratyphoid group were present but none of them were pathogenic for man. An additional study of 60 oxen, 63 pigs, 55 calves, and 15 sheep gave similar results.

Hogs under normal conditions probably harbor no organisms capable of causing food poisoning in man. Jordan examined 1,419 strains of organisms isolated from the intestines of 291 normal swine with entirely negative results. Diseased hogs may harbor at least three organisms, according to Jordan—*S. suipestifer, B. paratyphosus B.* (porcine), and *B. typhi suis* or *B. suipestifer-voldagsen.* Occasionally *S. enteriditis* is found.

Sheep in health harbor no food poisoning organisms. They are subject to infection with *Salmonella aertrycke,* however, and several European epidemics have been reported from such animals. The famous "mutton strain" was isolated from a man in Newcastle in 1911. Several epidemics of disease in sheep as well as food poisoning epidemics in man have been caused by organisms identical with the "mutton" type. Sheep as a rule are of less importance than cattle or hogs in food poisoning.

Rodents are extremely susceptible to infection with organisms of the Salmonella group. Friesleben (24) found 52 per cent of wild mice and 19 per cent of wild rats carrying such organisms in their intestines. Meyer (8) found 58 infected animals among 775 wild rats examined—28 with *S. enteriditis* and 30 with *S. aertrycke.* Zwick and Weichel found 28 out of 177 mice carrying organisms of this group. Price-Jones (25) fed enteriditis bacilli to rats, observing them in the liver and spleen in 48 hours. Two months later 10 per cent of the animals were carriers, while after five months a few animals still persisted as carriers. Such healthy carriers were capable of starting new epidemics among other rats. Guinea pigs have been found infected with an organism closely allied to *S. aertrycke,* but differentiated on a seriological basis, to which the name *S. pestis-caviae* has been applied. They likewise have been found infected with *S. aertrycke.* A few instances of infection in rabbits have been reported. Neither guinea pigs nor rabbits are relatively important compared to rats and mice.

Other animals harbor organisms of the Salmonella group but their relation to food poisoning is insignificant apparently. The horse is frequently infected with *S. abortus-equi;* the chicken with *S. pullorum* and *S. gallinarum;* the duck with *S. anatum.* None of these organisms have been incriminated in food poisoning epidemics. Torrey and Rahe (19) isolated *S. enteriditis* from a dog. Mori (20) as well as Krumwiede, Valentine, and Kohn (21), reported finding members of the group in cats. Jordan (9) has included in his collection of food poisoning organisms a culture of *S. suipestifer* isolated from the mesenteric gland of a monkey and a culture of *S. aertrycke* isolated from the spleen of a skunk.

Predisposing Factors to Food Poisoning

It has often been observed that more epidemics of food poisoning occur in hot weather than cold weather. Savage and White reported that 73.5 per cent of 222 epidemics in Great Britain occurred during the warm months of the year. In Prussia, 1920-23, 75 per cent of 2,250 outbreaks were in warm weather. It was formerly supposed that this was due to a more rapid multiplication of organisms in the contaminated food during the periods of higher temperature, which, without doubt, is a factor. Arnold (22) has recently drawn attention to the effect of high temperature and high humidity upon the susceptibility of the person, however. Young dogs show violent symptoms when fed enteriditis-infected meat and placed in a room with high humidity at

50° C., while under normal conditions the same animals are unaffected by such meat. Arnold agrees with Savage that enteriditis toxin is of importance. The autonomic nervous symptom is affected by the temperature and humidity combined, resulting in a decreased gastric acidity. He cites a food poisoning epidemic studied by Brophy (23) at Peoria, Illinois, where 60 per cent of 225 persons were involved. All the factors of high temperature, high humidity, worry, and hurry were present to disturb the normal functioning of the gastro-intestinal system.

Table 34

ORGANISMS OF THE SALMONELLA GROUP AND THEIR RELATION TO DISEASE IN MAN AND ANIMALS (After Savage).

Type	Disease in Man	Disease in animals
S. aertrycke.......	Food poisoning. Sporadic cases of illness unusual.	Cause of enteritis in rats, mice, guinea pigs and other rodents, and in parrots and other birds; occasionally a cause of calf enteritis.
S. enteriditis......	Gastro-enteritis of food poisoning type. Occasionally sporadic cases of illness.	Disease of cows and calves and rats.
S. suipestifer......	Paratyphoid fever, C. type with occasional food poisoning.	Secondary invader in pigs in hog cholera. Occasionally found as a primary cause of disease in pigs.
S. abortus—equi...	not found	Abortion of horses.
S. pullorum.......	not found	White diarrhea of chickens.
S. gallinarum.....	not found	Fowl typhoid of chickens.
S. psittacosis......	Atypical pneumonia	Enteritis of parrots.
S. paratyphi	Paratyphoid fever. Probably never produces food poisoning.	Not found.
S. schottmülleri....	Paratyphoid fever. Probably never produces food poisoning.	Not found.

Prevention of Food Poisoning

The prevention of food poisoning requires clean food that is handled as little as possible and consumed in as fresh a condition as possible. Animals suffering from any septic lesion are unfit for food unless it can be proven that the causative agent of the trouble is not a member of the Salmonella group. The emergency slaughter of sick animals is not generally practiced in the United States; careful inspection at regularly established abattoirs eliminates many diseased animals.

Food in markets and bakeries as well as in the home must be kept free from rodents which harbor numerous food poisoning organisms.

Food should be handled and manipulated as little as possible, especially food mixtures such as chopped meats, sausage, etc.

Foods prepared hours before the meal and allowed to stand are excellent culture media for the multiplication of enormous numbers of bacteria.

Foods may be rendered safe in many instances by thorough cooking. All organisms of the Salmonella group are thus readily killed while some of the toxins are destroyed. The toxin of the enteriditis bacillus is thermostabile, however, and its activity seems to be increased by heating.

ITEMS OF NOTE

1. Food poisoning (other than chemical poisoning and botulism) is not caused by decomposition of food products but by infection with members of the Salmonella group of organisms.
2. The Salmonella food poisoning organisms (other than paratyphoid A and paratyphoid B, which are of human origin) are entirely animal pathogens.
3. These organisms may produce intoxication within a few hours or infection later.
4. Meat from infected cattle, hogs, or sheep has been responsible for large numbers of outbreaks.
5. Wholesome food may be contaminated by infected hands or utensils or by droppings from infected rats and mice.
6. Predisposing factors to food poisoning are high temperature, high humidity, hurry, and worry.
7. The prevention of food poisoning calls for wholesome food kept clean, manipulated as little as possible, and eaten in as fresh condition as possible.

BIBLIOGRAPHY

1. GEIGER, J. C.
Jour. Am. Med. Asso., 1924, *81,* 1275.
2. ANON
Jour. Am. Med. Asso., 1928, *90,* 459.
3. SAVAGE, W. G., and WHITE, P. B.
Special Report Series, 91, Medical Research Council, 1925.

4. Bergey, D. H.
Manual of Determinative Bacteriology. Williams and Wilkins Co., Baltimore.

5. Klimmeck
Berlin Tierärzt. Woch., 1927, *43*, 613-22.

6. McWeeney, E. G.
Brit. Med. Jour., 1909, *1*, 1171.

7. Spray, R. S.
Jour. Am. Med. Asso., 1926, *86*, 109.

8. Meyer, K. F., and Matsummura, K.
Jour. Inf. Dis., 1927, *41*, 395.

9. Jordan, E. O.
J. Infect. Dis., 1925, *36*, 309.

10. Jordan, E. O.
Jour. Infec. Dis., 1923, *33*, 567.

11. Jordan, E. O.
The Bacteria of Food Poisoning. The Newer Knowledge of Bacteriology and Immunology, 1928, 445. Univ. of Chicago Press, Chicago.

12. Schütze, H.
Lancet, 1920, *1*, 93.

13. Salthe, O., and Krumwiede, C.
Am. Jour. Hyg., 1924, *4*, 23-31.

14. Winslow, C.-E. A., Hiscock, I. V., Rogers, O. F., and Robinson, E. S.
Am. Jour. Hyg., 1924, *3*, 328-246.

15. Rettger, L. F., Hull, T. G., and Sturges, W. S.
Jour. Exp. Med., 1916, *23*, 475-489.

16. Koser, S. A.
Jour. Inf. Dis., 1922, *31*, 79-88.

17. Damon, S. R., and Leiter, L. W.
Am. J. Hyg., 1927, *7*, 27-39.

18. Jordan, E. O.
Jour. Inf. Dis., 1918, *22*, 252.

19. Torrey, J. C., and Rahe, A. H.
Jour. Med. Res., 1912-13, *27*, 291.

20. Mori, N.
Centralbl. f. Bakt. I, Orig., 1905, *38*, 42.

21. Krumwiede, C., Valentine, E., and Kohn, L. A.
Jour. Med. Res., 1919, *39*, 449.

22. Arnold, L.
Illinois Med. Jour., 1928, *53*, 353.

23. Brophy, T. J.
Illinois Health News, 1926, *12*, 386.

24. Friesleben, M.
Deutsche med. Wchnschr., 1927, *53*, 1589.

25. Price-Jones, C.
Jour. Path. and Bact., 1927, *30,* 45-54.
26. Verder, Elizabeth
Am. Jour. of Public Health, 1927, *17,* 1007.
27. Savage, W. G., and Read, W. J.
Jour. of Hyg., 1913-14, *13,* 343.
28. Savage, W. G., and White, P. B.
Jour. of Hyg., 1922-23, *21,* 358.

CHAPTER XII

SWINE ERYSIPELAS

Swine erysipelas is a communicable disease of hogs caused by *Erysipelothrix rhusiopathiae;* man is susceptible to the disease and may be affected through skin abrasions.

HISTORY

Swine erysipelas probably has existed in Europe for many years, but it was confused with hog cholera and its history is not clear. It is said that it was first differentiated as a specific disease in France about 1846. Cartwright in England in 1847 described its chronic form without knowing its true nature. Pasteur in 1882 developed a vaccine for immunizing purposes by passing the virus through rabbits and then making broth cultures. Loeffler (11) in 1882 isolated the causative agent of the disease which he designated as *"Bacillus des Schweinerotlaufs."* Bang in 1888 and McFadyean in 1891 found the organism in chronic heart lesions of hogs.

PREVALENCE

The disease is prevalant in Europe, where it is known as "rouget" in France, "rotlauf" in Germany, and "erysipelas" in England. It exists to a considerable extent likewise in Austria, Denmark, Russia, Hungary, and other countries. In England in 1905, in a small strip of country four miles long by one mile broad, the disease attacked most of the hogs with 80 per cent mortality (1).

In the United States the existence of the disease had been suspected for many years. As early as 1885 Smith (12) isolated from hogs dead of hog cholera the closely allied mouse septicemia organism which has been confused with the swine erysipelas organism. Other investigators had similar experiences. In 1920 Ten Broeck (3) found organisms of this group in pigs dead of hog cholera, but failed to differentiate between the two species. It was generally believed therefore that swine erysipelas did not exist in the United States. In 1921 Creech (2) definitely established the fact that the disease did exist here by isolating *Ery-*

sipelothrix rhusiopathiae from several hogs which showed typical symptoms of "diamond skin disease."

The Causative Agent

Erysipelothrix rhusiopathiae (Bacillus erysipelatus suis, Bacillus rhusiopathiae suis) is a small organism 0.2 to 0.4 microns wide by 1 to 2 microns long, straight or only slightly bent. It is not motile and does not form spores. It is easily cultivated on the ordinary laboratory media.

While the organism is destroyed quickly by boiling water and within a few minutes by the usual disinfectants, it is very resistant to drying, in which state it will remain alive in the dark for a month or more and in the sunlight for 10 to 12 days. In salted and pickled meats it will remain alive 3 or 4 months.

A closely related organism is *Erysipelothrix murisepticae (Bacillus murisepticus* or *Pasteurella muriseptica)* producing a fatal septicemia in mice. It has occasionally been found in infected meat and has been confused with *Erysipelothrix rhusiopathiae.* When the swine erysipelas organism is injected immediately after isolation it is virulent for hogs, but when it has been grown on artificial culture media for any length of time, or after it has been passed through some of the experimental animals, it loses its virulence and then resembles the organism of mouse septicemia in that it fails to cause disease when injected into pigs. Kolda (6) found that when pure bile was used as a culture medium the virulence of *Erysipelathrix rhusiopathiae* was not decreased over a series of transplants, but when bile was mixed with other culture ingredients the virulence diminished.

The Disease in Animals

The disease is essentially an affection of pigs; it is seldom found naturally in other animals. The mouse and the pigeon are very susceptible to artificial injection, dying within three to five days; the rabbit is moderately susceptible to injection, exhibiting a local inflammatory lesion at the site of inoculation, but rarely dying; the guinea pig is very resistant. Horses, cattle, dogs, and cats are immune. Christiansen (7) reported an epidemic with high mortality among lambs, due to this organism. At the time of the outbreak there were no sick hogs in the vicinity. Poels found the organism in erysipelas of sheep. Schipp (8) in 1910 isolated from chickens suffering from enteritis, organisms which could not be distinguished from the swine erysipelas bacillus. Broll in 1911 recorded an outbreak in which all the fowls in one flock succumbed within a few weeks to this organism.

Pfaff (9) found chickens two to three months old fairly resistant in infection, but in chickens one to three weeks old, the rotlauf bacillus in pure culture caused an epidemic lasting two to five weeks. Jarosch described the organism as the cause of the disease in turkeys and Poels in the pigeon and fowl.

The disease in hogs occurs mostly in animals four to eight months old; young pigs under three months of age are fairly resistant as are also old hogs. This is only a relative immunity, however, and such animals become infected on some occasions.

Three forms of the disease have been observed—mild, acute, and chronic. The mild or urticarial form lasts only a few days, with great dullness, prostration, and high temperature, the diamond-shaped markings appearing on neck, back, and buttocks. Recovery is usually complete or with only a slight lameness. The acute form lasts from one to five days, with a sudden onset, extreme prostration, and extensive discoloring. The outcome is usually fatal. The chronic form usually is subsequent to a previous mild attack of the disease. There is first noticed an increased respiration with sometimes a cough, followed in a day or two by discoloration beginning at the tops of the ears and spreading along the ventral surface of the body. Death results in one to two weeks.

Infection is spread from hog to hog directly by ingestion. A considerable number of animals may be "carriers" harboring the organisms either in the intestinal tract or throat. Ten Broeck (3) isolated such bacilli from the tonsils of five out of sixteen pigs examined. Other workers in Europe previously had had similar experiences. Bauermeister in 1901 isolated the organism from the tonsils of 5 normal hogs out of 16 examined; Van Velzen in 1907 found it in the tonsils of 3 out of 11 pigs examined; Pitt (10) found it in 56 per cent of tonsils of 50 hogs, and in 40 per cent of glands of the ileocecal valve of 66 hogs.

The Disease in Man

Swine erysipelas is conveyed to man through abrasions of the skin. Infection by ingestion through food or water is doubtful and probably does not occur. The effects of the disease are not serious, the infection being confined to the site of inoculation in the form of an inflammatory lesion. Recovery takes place after 8 to 14 days.

Smith (4) reported 6 cases in men, all of whom had recently slaughtered infected hogs. Linser (5) described a case in a butcher who injured his hand while slaughtering a hog. Injection of 25 cc of swine erysipelas serum affected a cure in 3 days. Ruprecht states that the disease in man is quite prevalent in lower Bavaria. Gerlach in 1925 published an extensive article on the disease in man, stating that practically all cases came from contact with swine.

No cases in man have been reported in the United States.

Prevention

The prevention of swine erysipelas in hogs can be accomplished by vaccination. Pasteur originally used living organisms attenuated by passage through rabbits, two doses of the material being administered, the second of which was more virulent than the first. Sometimes, however, such living virus initiated the disease in healthy animals. The method has been modified, therefore, so that immune serum is given to the animal at the same time that the living culture is given. Immune serum is likewise efficacious in curing the disease if it is given early.

The prevention of the disease in man depends upon painstaking care by those persons who come into contact with infected hogs, that abrasions of the skin do not become contaminated.

Items of Note

1. Swine erysipelas is primarily a disease of hogs.
2. Few other animals are susceptible to infection.
3. Man is infected through abrasions of the skin and probably not by ingestion.
4. The prevention of the disease in hogs may be accomplished by vaccination plus immune serum.
5. The prevention of the disease in man requires precaution as to cuts and abrasions by those who come into contact with hogs.

BIBLIOGRAPHY

1. HOARE, E. W.
   A System of Veterinary Medicine. Alex. Eger, Chicago, 1915.
2. CREECH, G. T.
   Jour. Am. Vet. Med. Asso. 1921, *59*, 139-150.
3. TEN BROECK, C.
   Jour. Exp. Med. 1920, *32*, 331.
4. SMITH, N.
   Abs. Jour. Am. Med. Asso., 1918, *70*, 1200.
5. LINSER,
   Dermatal. Zeitschr, 1911, *18*, 352-354.
6. KOLDA, J.
   Rev. Gen. Med. Vet., 1924, *33*, 293-312.
7. CHRISTIANSEN, M.
   Abs. Exp. Stat. Rep., 1921, *44*, 583.
8. SCHIPP, C.
   Deutsch. tierartzl. Woch., 1910, *18*, 97.
9. PFAFF, F.
   Abs. Exp. Stat. Rep., 1923, *49*, 81.
10. PITT, W.
    Centralbl. f. Bakt. 1 Orig., 1907, *45*, 33, 111.
11. LOEFFLER
    Arb. aus dem Kaiserl. Gesundheits, 1885, *1*, 46.
12. SMITH, T.
    U. S. Dep. Agr., Bureau Animal Indus. 2nd Annual Report, 1886, 196.

CHAPTER XIII

AFFECTIONS CAUSED BY ANIMAL PARASITES

TRICHINELLA SPIRALIS

Trichinosis is primarily a disease of animals, principle among which figure the hog and the rat. Man is extremely susceptible and may become infected from eating trichinous pork.

The disease is probably of ancient origin. The Hebrew prohibition against pork, promulgated by Moses about 1500 B.C., was without doubt due, in part at least, to the danger of trichinosis. The first definite information concerning the affection appeared in 1822, when Teidemann in Germany observed encysted forms of the organisms in muscle tissue. In 1828 Peacock in England not only observed the parasite but prepared a specimen which is still in existence at Guy's Museum in London (1). The parasitic nature of the disease was suggested by Hilton in 1832, which led to the discovery of the embryo by Owen in 1835, with the designation *Trichina spiralis.* During the next fifty years much work was done on the life cycle of the parasite by various investigators—Siebold 1844, Dujardin 1845, Herbst 1845, Leidy 1847, Zenker 1860. It was Virchow, however, who first described the life cycle as it is now known.

Trichinella spiralis is a round worm which, with *Trichuris trichuria,* belongs to the *Superfamily Trichuroidea.* The adult female trichinae are three to four millimeters long, scarcely visible to the naked eye—white in color, slender, and tapering towards the anterior end. The adult males are about half as large. This is the stage that lives free in the intestine. The embryos when born are scarcely more than 0.1 mm. long, but after the migration from the intestine through the lymphatics and blood stream to the destination in the muscles they grow to a length of 0.8 to 1.0 millimeter. The cysts consist of one or two or more embryos which have become sexually differentiated and coiled up within a calcareous covering. They are lemon-shaped, from 0.25 to 0.5 millimeters long, lying parallel with the muscle fibers.

The life cycle of the parasite begins when meat is ingested in which the cysts are present. The gastric juices dissolve the cysts and set free the worms. They pass into the intestine where they develop into adults, male and female, in a period of about four days. After copulation, the males pass out of the intestine and die. The females begin discharging embryos within a few days and continue the process for a month or more, after which they also disappear. The embryos penetrate the intestinal wall, entering the lymphatics and blood stream where they wander to different parts of the body, becoming located in the muscles after a week or ten days. The parasites become encysted in the muscle, retaining their vitality for a year or two, although cases are reported of worms being alive eleven years after infestation. If the muscle is ingested by another animal before the worms are dead, the cycle starts over again.

Trichinosis in Animals

The reservoir of infection of trichina is the hog and the rat. Either animal may contract the affection from the other, but each is capable of harboring the parasite for its entire life cycle.

About 1880 American pork was so commonly infected with trichina that it was excluded from German markets. The result of the investigation which followed was the beginning of modern meat inspection in the United States.

From 1898 to 1906 the United States Department of Agriculture made microscopic examinations on specimens of tissue from 8,257,928 hogs, of which 115,812 showed living trichina—1.41 per cent. The rate, without doubt, was higher than this, since microscopic examination fails to detect all specimens harboring the parasites.

In Germany in 1885 the infection in hogs was 5.5 per 10,000, but by 1899 this was reduced to 1.4 per 10,000. In different parts of the country, however, the rate varied in 1896 from 27.2 per 10,000 in Posen to 2.1 in Prussia as a whole; by 1906 the Posen rate had fallen to 6.1 and the Prussian to 0.5 per 10,000.

Rats are responsible for propagating and spreading trichinosis in many instances. It is almost impossible to eliminate rats entirely around hog pens and abattoirs or prevent them from migrating to new fields. The common brown rat, as well as the roof rat and black rat, are easily infected. In Boston 10 per cent of rats caught around slaughter houses were found to harbor trichina, but this rate rose to 76 per cent in rats caught around knackers' yards where the offal from slaughter houses was handled. In

Saxony and Austria a similar observation was made, the rate being 8.3 per cent among the slaughter house rats, rising to 22.1 per cent around knackers' yards. Bovaird (2) estimates that 2.3 per cent of rats around slaughter houses ordinarily harbor trichina parasites.

Experimental infection of the laboratory animals, such as guinea pigs and rabbits, is easily accomplished. Natural infection has been noted in wild animals such as the bear, badger, fox, and mouse. The dog has sometimes been found to show infection but this is difficult to demonstrate artificially. Cats rarely show infection. Horses, cattle, and sheep are quite refactory with the development of the worms in the intestine only and no encystment in the muscles. The same is true of such birds as the pigeon, goose, chicken, and grouse.

Trichinosis as an agricultural problem has received little attention in the past few years. In the Agricultural Index for seven years, 1921 to 1927, but two references are listed, one dealing with the effects of pork curing processes on trichinae (3) and one on the effect of feeding dead trichinae to animals (4).

Trichinosis in Man

Trichinosis in man is confined largely to those countries where pork or pork products are eaten raw or insufficiently cooked. Thus the rates among the Germans, Italians, and Austrians in the past have been much higher than among the English and French because of the difference in food habits.

The extent of infection in the United States is not great. Bovaird estimated that about 1,200 cases had occurred in the country up to 1917. Ransom (5) gives a somewhat higher figure, listing 1,558 cases occurring between 1842 and 1914; all parts of the country were affected, but a preponderance of cases appeared in New York State and Pennsylvania because of the larger numbers of foreign population. Numerous small outbreaks have occurred but no large epidemics. In 1882 in Minnesota there were 15 cases and 1 death; in Massachusetts 50 cases and 4 deaths; in 1911 in California 58 cases and 1 death; in 1915 in Wisconsin 21 cases and 3 deaths; in 1917 in New York State 14 cases (6); in 1923 in Connecticut 35 cases with 9 other sporadic infections (7); in 1928 in New York State 43 cases (1). Many other smaller outbreaks are recorded in the medical literature. There are no exact figures, however, on the number of human infections of trichinosis. If the infection is slight, there may be no symptoms at all, or only symptoms so indefinite as to be overlooked. Four cases of trichinosis occurred among university students in Michigan in 1924 who had attended a football game in Illinois and had eaten insufficiently cooked pork at a certain restaurant. Although hundreds of other persons ate at the same restaurant, no other cases of infection could be found. Bettison (8) considers that "it is logical to assume that many others probably were infected with the parasite, but were so mildly affected that their condition either was not recognized or did not come to the attention of a physician." Severe cases may be mistaken for typhoid fever or for chronic rheumatism. There is not even complete and systematic reporting to health authorities of cases which have been diagnosed. Osler estimated the percentage of infection as 0.6 in the general population on the basis of 1,000 post-mortem examinations. Williams (9) in 1901 examined 505 bodies at autopsy, finding parasites in 27 or 5.34 per cent. These persons were mostly from institutions or almshouses where the rate of infection might naturally be higher than in the general population.

The death rate is not high in trichinosis, ranging from 5 per cent to 15 per cent in most studies. Ransom found 19 deaths among 320 cases in the United States between 1909-14—6 per cent mortality; among 1,550 cases which he studied later there were 270 deaths—16 per cent mortality. Among 6,329 cases in Germany between 1881 and 1898 there were 318 deaths—5 per cent mortality. In all of these instances, however, it was only the more severe cases that were studied, while numerous mild cases went unrecognized.

The symptoms of infection in man are not constant and sometimes are entirely lacking. During the first week after ingestion of the parasites—the invasion period, when the worms are developing in the intestine—there may be loss of appetite, nausea, vomiting, and diarrhea. The second period, that of dissemination, begins between the ninth and fifteenth days; the embryos attack the muscle walls causing severe muscular tenderness; fever is present; pulmonary symptoms are common. The third period, known as the period of encystment or regression, lasts until the patient recovers; the symptoms are severe with edema of the eyelids, sweating, and increased temperature.

Trichinosis is often mistaken for other infections—typhoid fever, meningitis, frontal sinusitis, dermatomyositis. Laboratory examinations will differentiate it, however. The most common finding is an eosinophilia ranging from 15 to 60 per cent or higher. At times the embryos may be found in the intestine; during the encysting stage the worms may be demonstrated in the tissue. Bachman (10) has described a precipitin test, using the patient's blood serum with an antigen composed of dried trichinella powder. An infected person gives positive results in dilutions as high as 1:4000. More recently Bachman (11) has used a solution of the dried trichinella powder for intracutaneous injections, obtaing typical allergic reactions in animals within the first week of the disease, whereas the precipitin test is not positive until three or four weeks have elapsed.

There is no known remedy for the affection which is specific. Control of the disease therefore depends upon prevention.

Prevention of Trichinosis

It has been shown by Raffensperger (12) that trichinae are not transmissable through the feces, that unencysted trichinæ are not capable of development when meat containing them is ingested and by Schwartz (4) that dead or non-viable trichinae produce no ill effects even when ingested in large numbers. The prevention of infection of man, therefore, depends upon the prevention of ingesting living, encysted forms. Four ways suggest themselves to accomplish this.

1. Prevention of infection of hogs. This is an agricultural problem difficult of solution. It calls for destruction of rats in and around hog pens. Hogs should not be fed on slaughter house offal, nor be allowed to consume other dead hogs or dead rats. Because the affection in hogs is difficult to determine either antemorten or postmortem, ordinary precautions are apt to be omitted.

2. Inspection of Slaughter Houses. No satisfactory method of inspection has yet been devised to detect the presence of trichinae. Hogs show no characteristic symptoms during life, and no gross pathological lesions after death. In 1898 microscopic examinations of all pork intended for export to other countries was commenced. Small pieces of muscle pressed between two pieces of glass and examined under the low power of the microscope sometimes revealed the encysted larvae. The failures were so high, however, that the practice was discontinued in 1906. In Germany between 1881 and 1898 Stiles studied 6,329 cases of trichina, of which 2,042, or 32 per cent, were traced to pork which had been inspected by this method and passed as free from infection.

At the present time federal meat inspection regulations include no inspection for this disease.

3. Destruction of parasites by refrigeration. Trichinae are quite susceptible to cold. Ransom (13) showed that above 10° F. the organisms will survive for a long time in meat. Only a few survive when kept at 5° F. (—15° C.) for five days. Different strains of the organism vary in susceptibility to cold. Storage of pork at a temperature of 5° F. or below for twenty days destroys all trichinae with a safety margin of about ten days. The United States Department of Agriculture requires such storage for all pork killed at slaughter houses under federal inspection. Variations are allowed in certain instances for sausage or other pork products, such as salting and drying. The salt draws the water from the parasite, thus lowering its vitality as well as making it more susceptible to drying.

4. Cooking. The United States Department of Agriculture warns all persons "not to eat pork, or sausage containing pork, whether it has been officially inspected or not, until it has been properly cooked." Proper cooking renders pork perfectly safe, even though it contains living parasites. Ransom (14) showed that a temperature of 55° C. quickly destroys the organism. At temperatures below this the larvae may recover, but they die if heated for a long time, depending upon coagulation of protoplasm. Heat at 50° C. applied for a long time destroys the organism by exhaustion following excessive activity to which it is stimulated by the heat. The regulations for all plants operating under Federal inspection require a temperature for meat cooked in the establishments of 137° F. (58.3° C.) as a minimum, which gives a satisfactory margin of safety. Since there are numerous slaughter houses not under federal supervision, and the above precautions are not carried out, proper cooking in the home must be resorted to as a complete safeguard.

Ascaris Lumbricoides

Besides Trichinella spiralis, the only other round worm of significance is Ascaris lumbricoides. This is the so-called eelworm, one of the commonest of human parasites. It occurs in all parts of the world, especially in children, being abundant in the tropics. It is one of the largest of the round worm parasites. The females average ten inches or more in length and about a quarter of an inch in diameter while the males are shorter. The eggs, deposited by the female in enormous numbers in the intestine of the host, are passed out with the dejecta where they "ripen" in two to six weeks according to the temperature and nature of the soil (16). When such eggs are ingested by man, or other animals, they pass to the small intestine where the eggs hatch. The larvæ penetrate through the walls of the intestine into the blood vessels and are carried to the liver, then to the lungs where they break out into the air passages. They then migrate to the mouth and are swallowed. During this time they are developing slowly and when they again reach the intestine they continue to develop into the adult worm provided they are in the proper host. Sometimes the adult worms are found in the bile ducts or pancreas or even the larynx and eustachian tube, where they produce serious complications. In the tropics it is necessary to take them into consideration in cases presenting symptoms of intestinal obstruction or appendicitis.

Ascaris lumbricoides, the parasite of man, may be identical with Ascaris suis, the parasite of the hog. Caldwell (15) made a survey in Alabama in which epidemiological evidence was obtained indicating that the pig and human Ascaris were not identical. In an area in which the incidence of Ascaris infestation of pigs was 46.5 per cent and in which all factors seemed to be favorable for cross infestation between pig and man, the incidence of human Ascaris was less than 1 per cent.

The intensity of Ascaris infection in children is five times that in adults (17). This is partly due to an increased susceptibility of children and partly to more careless habits. In warmer climates the rates are higher. Among 500 Filipino children, varying in age from 8 to 18, Lissner (18) found in a single stool examination from each one a rate of 73.0 per cent. In Philadelphia de Rivas and Fife (19) found a rate of 1.5 per cent among 512 hospital patients and 2.4 among 312 private patients.

Prevention of Ascaris infestations depends upon the development of a "sanitary conscience," which is almost entirely lacking in children. Fortunately oil of chenopodium is very efficient in expelling the worms.

TAPEWORMS

There are several tapeworms which man shares with the lower animals. The beef tapeworm spends its larval stage in the muscular tissue of cattle, but enjoys the hospitality of the human intestine for its adult stage. A similar relationship exists between man and swine for the pork tapeworm, and between man and fish for the fish tapeworm.

The tapeworms belong to the cestode class of the Platyhelminthes or flat worms. Their parasitic life has developed to such an extent that with the exception of the fish tapeworm they are incapable of carrying on any part of their life cycle independent of a host. The adults have entirely lost all trace of a digestive tract and absorb food from the intestine of the host all over the surface of their bodies.

All the tapeworms have a somewhat similar life history. The adult form, existing in the intestine of its proper host, consists of a "head" or scolex and numerous segments or proglottids. The latter are self-fertilizing and contain enormous numbers of eggs. Some species develop the embryo in the proglottid. As the proglottids become "ripe" they are broken off from the tapeworm and discharged from the intestine; each egg or embryo is enclosed in a tough encasement capable of protecting it for a long period of time. When eaten by another host of the proper species, the tough shell is dissolved off. The embryo passes into the organs and tissues of the new host and is carried by lymph and blood streams to selective tissues or to all parts of the body according to the species of parasite. Further development takes place within the tissue to the larvæ or bladder worm stage, at which time the "head" or scolex, with its suckers, is formed. In this stage the parasites remain until the tissue is ingested by a new host. All but the head is digested off, and this attaches itself by means of its suckers to the wall of the intestine, where it proceeds to develop new proglottids.

Beef Tapeworm

The beef tapeworm, *Taenia saginata,* is the commonest human tapeworm. It occurs in all parts of the world. Although the head of an adult is scarcely larger than a pin head, it may develop nearly two thousand proglottids, extending to a length of fifteen or twenty feet or more. An infected man, therefore, expels many thousands of eggs. One of these eggs, carried to the drinking water or to the food of a bovine or other ruminant by any accidental means, develops in the muscle of the new host to the larval stage. Because the cysts present no contrast in appearance to the surrounding muscle tissue, their presence is easily overlooked. In meat inspection the muscles of the heart or the tongue are examined, for these present the parasites in the most abundance.

Beef containing bladder-worms is said to be "measly." Man may become infected by eating such meat raw or partially cooked. The adult stage of the parasite develops in man in about two months.

The prevention of the disease consists first in careful disposal of human excreta so that cattle may not become infected and second in proper cooking of beef to destroy the bladder worms. Refrigeration likewise destroys the parasite within a few weeks.

The Pork Tapeworm

The pork tapeworn, *Taenia solium,* is fairly common in some portions of the globe but rare in the United States. The adult has less than a thousand proglottids, which are discharged from man one at a time. Encysted eggs which may be ingested by hogs, develop into larvæ and localize in the shoulder muscles, neck or tongue. Such measly pork will cause human infection when eaten raw or improperly cooked. As with the case of measly beef, prevention consists in proper disposal of human excrement and proper cooking of pork. Unlike the beef tapeworm however, the pork tapeworm is capable of establishing the larval stage in man if an egg should be ingested. The embryos tend to invade the brain or the eye resulting in convulsions, blindness or death.

The bladder stage of the parasite can likewise be passed in the dog and possibly some other animals.

The Fish Tapeworm

The fish tapeworm, *Diphyllobothrium latum,* was originally a native of eastern Europe, but in recent years it has become a common inhabitant of the large lakes of Canada and the northern central states of the United States. There is little doubt that these waters were free from such parasites in the early days, but were contaminated by infested European immigrants settling in the region—Scandinavian, Finnish, Russian and German. Magath (20) found in Minnesota that only the lakes near the large towns were infested and the distant lakes were free. He argued that if the broad tapeworm had existed in this region before the settlement of the Europeans the reverse would have been true. Vergeer (21) found similar conditions in Canada but discovered that in addition to persons, dogs were an important reservoir of this tapeworm. Indians and others feed their dogs on fish, and the dogs so infected may harbor from five to nineteen broad tapeworms.

McGavran and Songkla (22) list eleven cases of this infection occurring in native born Americans residing in Minnesota, Michigan, Illinois and Indiana, and add two more cases in Boston. An investigation of the supply of fresh water fish in the Boston market indicated that it came largely from the Great Lakes region. Because of the wide market distribution of these fish, the infection may be more widespread in this country than realized. Warthin (23) at the University of Michigan states that whereas his laboratory formerly came into contact with two or three cases a year, this incidence has now doubled or trebled.

The adult worm as it lives in man has from two thousand to four thousand short broad proglottids, extending in its entire length thirty feet or more. The eggs are voided from the proglottids before the latter break off and are passed from the intestine singly. Each egg hatches out in water, where the larvæ develop. These are ingested by fish, where the bladder forms become encysted in the muscles in the course of one or two weeks. Such fish, when insufficiently cooked, will infect man.

The Dog Tapeworm

The dog tapeworm *Taenia echinococcus,* is fairly common in Southeastern United States. In Iceland, Australia and some other countries the infection is very prevalent. The adult worm lives in the intestine of the dog. It has only three or four segments, each containing several hundred eggs.

Man contracts the disease through association with the dog. Egg-bearing segments eliminated from the intestine of the dog, finding their way to the mouth of man, develop the embryos in his stomach. They pass by a selective action into the liver, though sometimes into the lungs, kidneys, spleen or other organs. Cysts form slowly and at times reach six inches in diameter.

Sheep, cattle and other herbivorous animals are capable of harboring the larval form of the worm. Dogs which eat raw meat from such animals, as around slaughter houses, become infected, thus completing the life cycle of the parasite. It is in this way that the infection is kept going.

Prevention consists in avoidance of close familiarity with dogs, especially "kissing." Dogs should not be allowed to infect either food or drinking water. The prevention of access of dogs to the surroundings of slaughter houses would tend to reduce infection in them.

Dipylidium caninum in its larval stage is transferred from dogs and cats by way of infected fleas to spend its adult days in the intestine of children.

FLUKES

Flukes. The flukes which are transmitted from animal to man are of small importance in the United States. The liver fluke of sheep *Fasciola hepatica,* is occasionally found in man. In the Orient *Clonorchis sinensis,* the Chinese fluke, is rather common in cats, dogs, and man. In Europe *Opisthorchis felineus* often occurs in man as well as dogs and cats. Various other liver flukes are occasionally transmitted to man from fish and snails.

Several intestinal flukes are normal inhabitants of animals but may also infect man especially in the Orient. Among the most common is *Fasciolopsis* of hogs. *Paramphistomum cervi* is found in the stomachs of cattle in Egypt while a closely related species is found in the United States. The parasite is not of consequence in the latter country in man however.

ITEMS OF NOTE

1. Trichinosis, caused by *Trichinella spiralis,* is primarily an affection of hogs and rats.
2. The infection of man is the result of accidental ingestion of the encysted larvæ in trichinous pork.
3. *Ascaris lumbricoides,* the cause of ascariasis, finds its host in hog and man.
4. The infection of man takes place by the accidental ingestion of ascaris eggs through infected drinking water or otherwise.

5. The beef tapeworm, *Taenia saginata,* may reach the human intestine through the medium of insufficiently cooked beef.
6. The pork tapeworm, *Taenia solium,* may establish its residence in man either through insufficiently cooked pork or by ingestion of the eggs through contaminated drinking water, etc.
7. The fish tapeworm, *Diphyllobothrium latum,* infests man as the result of the ingestion of insufficiently cooked fish.
8. The dog tapeworm *Taenia echinococcus,* is transmitted to man directly through association with dogs, whereby the eggs from the intestine of the dog find their way to the mouth of man.
9. The flukes that may be transmitted from animals to man are of small importance in the United States.
10. Transmission to man of the parasites which are harbored by the lower animals may be prevented by thorough cooking of the meat of such animals in the case of *Trichinella spiralis, Taenia saginata, Taenia solium, Diphyllobothrium latum,* and certain of the flukes. Precautions against the ingestion of the eggs of the parasites through drinking water or contaminated fingers are necessary in the case of *Ascaris lumbricoides, Taenia solium, Taenia echinococcus* and certain of the flukes.

No attempt is made to discuss certain tropical diseases, such as sleeping sickness, trypanosomiasis and the like, several of which are caused by animal parasites and are transmitted by means of insects.

BIBLIOGRAPHY

1. McDonald, Edward P., and Waddell, Kenneth C.
Jour. Am. Med. Asso., 1929, *92,* 449.
2. Bovaird, David, Jr.
Med. Rec. 1917, *91,* 261-264.
3. Ranson, H. B., and Schwartz, B.
United States Dept. of Agriculture Bull. 880 Sept. 10, 1920.
4. Schwartz, Benjamin
Jour. Agri. Res., 1925, *31,* 945-947.
5. Ranson, H. B.
United States Live Stock Sanitary Asso., Report of 18th Annual Meeting, p. 147.
6. Salzer, B. P.
Med. Record, 1917, *91,* 261.

7. Alexander, M. E.
Amer. Jour. Med. Sci., 1923, *165,* 567-577.
8. Bettison, Wm. L.
Jour. Am. Med. Asso., 1925, *86,* 609.
9. Williams, H. U.
Jour. Med. Res., 1901, *6,* 64.
10. Bachman, George W.
Jour. Prev. Med., 1928, *2,* 35-48.
11. Bachman, George W.
Jour. Prev. Med., 1928, *2,* 513-523.
12. Raffensperger, H. B.
Jour. Am. Vet. Med. Asso., 1918, *53,* 363-367.
13. Ransom, H. B.
Jour. Agri. Res., 1916, *5,* 819-854.
14. Ransom, H. B.
Jour. Agri. Res., 1919, *17,* 201-221.
15. Caldwell, F. C., and E. L.
Jour. Parasitol, 1926, *13,* 141-145.
16. Brown, H. W.
Jour. Parasitol, 1927, *14,* 1-15.
17. Child Health Bulletin, 1928, *4,* 50.
18. Lissner, L.
Münch. Med. Wochnschr, 1928, *75,* 1163.
19. de Rivas, Damaso, and Fife, Charles A.
Jour. Am. Med. Asso., 1929, *92,* 624-627.
20. Magath, T. B.
Minnesota Med., 1927, *10,* 614-616.
21. Vergeer, Teunis
Jour. Am. Med. Asso., 1929, *92,* 607-608.
22. McGavran, E. G., and Songkla, Mahidol
Jour. Am. Med. Asso., 1928, *90,* 1607-1608.
23. Warthin, A. S.
Jour. Am. Med. Asso., 1928, *90,* 2080-2082.

Section Two

RODENT AFFECTIONS

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CHAPTER XIV

PLAGUE

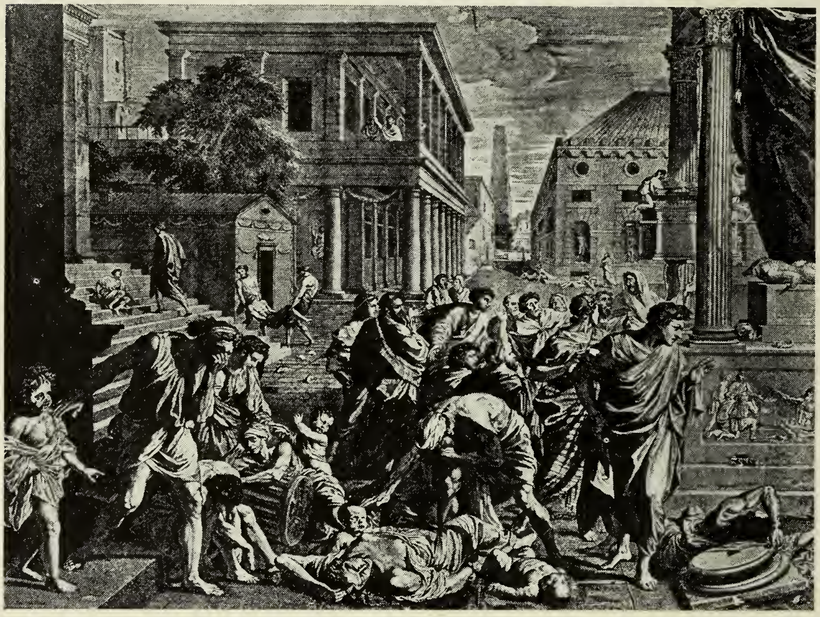
Plague is an acute infectious disease caused by *Pasteurella pestis.* Primarily it is a disease of rodents, especially rats; secondarily it is a disease of man. The bubonic type in man is transmitted from rodents by means of infected fleas; the pneumonic type spreads from man to man directly.

HISTORY

During the 1500 years before the Christian era there were forty-one epidemics of plague (16). One of the early records of its ravages, is recounted in the Bible (1 Samuel, Chapters 5 and 6). The Philistines made golden images of the "mice" that over-ran the land, in order to stay the pestilence. The outbreak began at the seaport town of Ashdad and moved inland. At Beth-shemesh the mortality is given as 50,070.

Outbreaks occurred from time to time during the ensuing centuries but records sufficiently accurate were not kept to make even the diagnosis certain. Papon (16) however, states there were 109 epidemics during the first 1500 years of the Christian era and 45 from 1500 to 1720.

The first authentic epidemic of plague began in 542 A.D. at Pelusium in Egypt. This city was a leading center for trade between the East and West. The main trades routes at that time between Europe and the East were along the Mediterranean Sea and overland through Turkey, Germany, and France. It was along these routes that the infection slowly spread till every country then known was afflicted, from Asia to Ireland. At the height of the epidemic the mortality was 5,000 persons daily, and at times this reached 10,000. According to Procopius, "it spared neither island nor cave nor mountain top where man dwelt. . . . . Many houses were left empty and it came to pass that many from want of relatives or servants lay unburied for several days. At that time it was hard to find anyone at business in Byzantium. Most people who met in the streets were bearing a corpse. All business had ceased. All craftsmen had deserted their crafts." After about two hundred years the epidemic subsided.

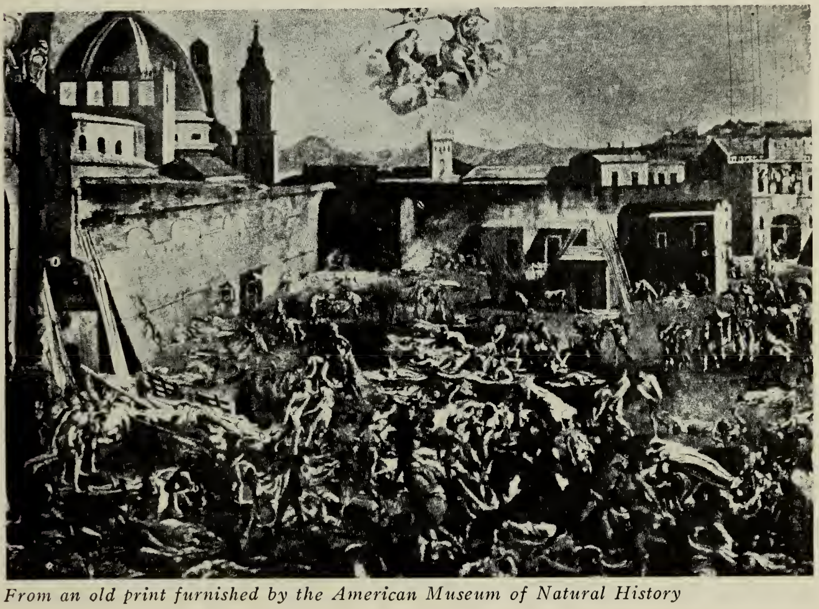


Print from the American Museum of Natural History

FIG. 19.—Plague among the Philistines. A 17th Century conception as depicted by Nicolas Poussin. Although it was nearly three hundred years later that the relation of the rat to plague was definitely established, the presence of large numbers of rats is here shown.

The second pandemic of plague, known as the Black Death, originated in Mesopotamia about the middle of the eleventh century, attaining its height in the fourteenth century and not disappearing till the close of the seventeenth century. It is thought that the crusaders, returning from the Holy Land in the twelfth and thirteenth centuries, were instrumental in hastening the spread of the disease. Trade routes were primarily involved, however, the infection spreading to the East, the West and the North over the then civilized world. During the course of the disease one-fourth of the world's population—25,000,000 people—perished from it. Towns were left empty and all trade was at an end, for none knew when their turn would come to be smitten. In London in 1664 and 1665 in a population of 500,000, there were 70,000 deaths from plague.

The chief factor in the subsidence of this epidemic was the discovery of a new trade route between the countries of northern Europe and India by way of the Cape of Good Hope, thus eliminating the endemic centers of Asia Minor. The disease disappeared soon after this, except for limited outbreaks occurring from time to time during the next few centuries.



From an old print furnished by the American Museum of Natural History

Fig. 20.—An ancient conception of the cause of plague, showing the angel of death wreaking vengeance on the populace.

The last great pandemic began in 1871 and has not yet completely subsided. It originated in the town of Junan Fu in China, spreading slowly over China and India and thence by trades routes over the world. There was scarcely a country but the infection appeared at its seaports. In 1907 the epidemic was at its height, the disease occuring in Persia, China, Japan, India, Russia, Indo-China, the Philippines, Arabia, Egypt, Tunisia, Algeria, West Africa, South Africa, East Africa, Scotland, Argentina, Brazil, Chili, Paraguay, Peru, Uruguay, Australia, New Zealand and the United States. In most countries modern science prevented the spread of the infection beyond the seaport towns. In the United States the ground squirrels of California became infected and have acted as a focus of infection, a few cases in man appearing there almost every year since 1903.

In India, according to the Ministry of Health of Great Britain, ten and a half million people died of plague between 1898 and 1923. The League of Nations Health Organization presents the following mortality table for 25 years:

PLAGUE MORTALITY IN INDIA
1898-1923

1898-1903	1,707,456 deaths
1903-1908	4,325,237 deaths
1908-1913	2,042,127 deaths
1913-1918	2,179,401 deaths
1918-1923	530,170 deaths

In China where the epidemic had spread rapidly, with grave results, Hong Kong was infected in 1894. In June of that year Kitasato (11), working in Hong Kong, isolated the plague bacillus; while a little later in the same city Yersin (12), working independently, duplicated his results.

Several countries established plague commissions—Austria, Germany and Russia—which did excellent work. The last Indian Commission has given us much of our knowledge concerning the epidemiology of the disease.

The Infectious Agent

Pasteurella pestis, commonly known as *Bacillus pestis,* belongs to the hemorrhagic septicemia group. It is a small rod, 0.5 to 0.7 microns wide by 1.5 to 2.0 microns long, usually occurring singly, but sometimes in pairs or short chains. It is quite pleomorphic, appearing in tissues or abscesses as a rod with rounded ends resembling an oval form; in smears from old buboes, club shaped forms are found; at other times yeast like forms are present. Growth takes place easily upon ordinary culture media. Since no spores are formed, it is easily killed by the usual disinfectants.

Table 35

HUMAN CASES OF BUBONIC PLAGUE IN THE UNITED STATES

Year	California	Louisiana	Texas	Florida	Washington (State)	Total
1900	22	..	..	..	..	22
1901	26	..	..	..	..	26
1902	35	..	..	..	..	35
1903	17	..	..	..	..	17
1904	10	..	..	..	..	10
1907	170	..	..	..	7	177
1908	7	..	..	..	..	7
1909	1	..	..	..	..	1
1910	2	..	..	..	..	2
1911	3	..	..	..	..	3
1913	2	..	..	..	1	3
1914	..	30	..	..	..	30
1915	1	1	..	..	..	2
1916	..	..	..	..	..	0
1917	..	..	..	..	..	0
1918	..	..	..	..	..	0
1919	1	12	..	..	..	13
1920	1	7	31	10	..	49
1921	1	..	..	..	..	1
1922	1	..	..	..	..	1
1923	1	..	..	..	..	1
1924	41	..	..	..	..	41
1925	..	..	..	..	..	0
1926	..	..	..	..	..	0

Animals Susceptible

The highly susceptible animals include man, monkeys, rats, mice, squirrels, prairie dogs, woodchucks, weasels, gophers, tarbagans, guinea pigs and rabbits.

Cats, camels and kangaroos are somewhat susceptible, having been found naturally infected. Horses and llamas have been infected by the injection of large doses of the organisms but never have been found naturally infected. Donkeys are resistant to natural infection except under extreme conditions.

Cattle, sheep, pigs, dogs, chickens, geese, reptiles, and probably bats are resistant to infection with the pestis bacillus.

Geographical Prevalence

According to Robertson of the United States Public Health Service, "Bubonic plague is essentially a disease of hot climates and having been introduced into tropical countries, it tends to persist indefinitely. Outside the immediate tropics, this disease is rather definitely limited in the extent to which it will spread.

Distribution of Plague throughout the World in 1926.

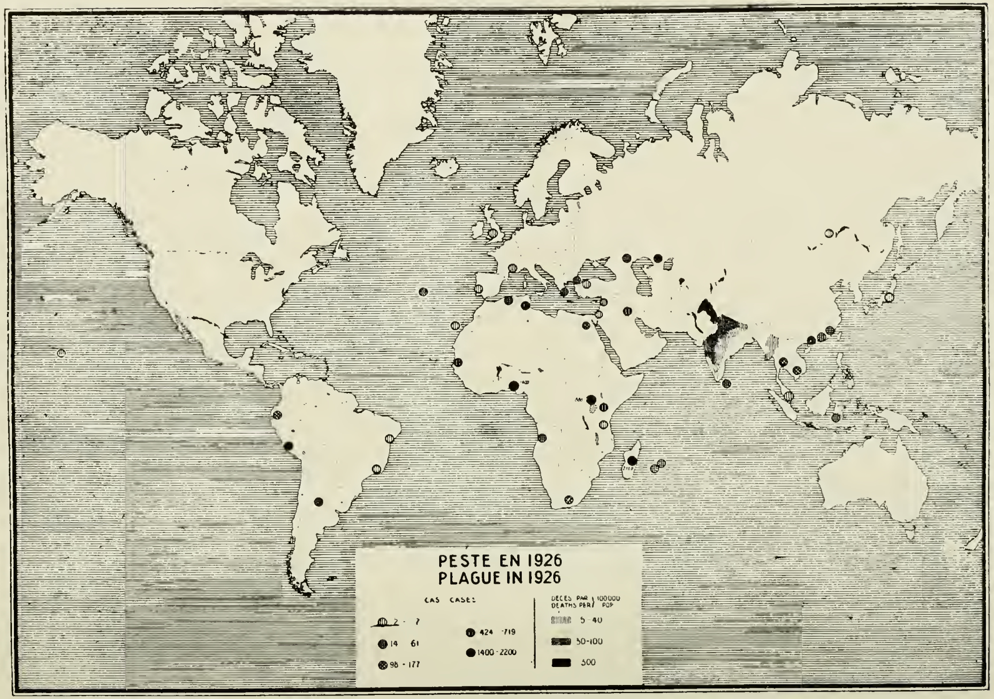

PESTE EN 1926
PLAGUE IN 1926
CAS CASES
2 - 7
14 61
98 - 177
424 -719
1400 - 2200
DECES PAR } 100000
DEATHS PER } POP
5 - 40
50-100
500

Fig. 21.—From the Fourth Epidemiological Report of the League of Nations.

"In countries with a mean midwinter temperature of 45°F. or below, bubonic plague is occasional, accidental and distinctly self-limited, and it seems possible for it to occur in the colder regions only for short periods under unusual conditions."

Central Asia is reputed to be the original home, not only of the human race, but also of plague. That area has been a perpetual focus of infection and from Asia bubonic plague has followed man in his trade and commerce to almost every country in the world. All of the pandemics have been traced to that source. Other foci of infection have appeared, however, which are potential sources of danger. Wu Lien Teh has listed such endemic foci as follows (1):

Africa	Earliest known infection
Benghazi	1270
Central Africa	1864
Asia	
Arabia (Assyr)	1157
Kurdistan	oldest records.
India (Kumaon)	1823
China (Yunan)	1853
Mongolia	1856
Thibet	1900
Turkestan	1884
Persia	1876
Astrakhan	1364

The most important areas, as far as serious outbreaks are concerned, are Kurdistan and the region around the Himalayas, Thibet, Yunnan and Kumaon.

In the United States plague has been endemic in the ground squirrels of California since 1903, with occasional cases almost every year in man. In San Francisco in 1900, and afterwards for three or four years, human plague was undoubtedly due to rats.

Seasonal Prevalence

The seasonal prevalence of the bubonic type of plague depends largely upon the influence of the weather on the breeding of rats and rat fleas. The Indian Commission found that in temperate regions outbreaks occurred chiefly during the summer months. In hot, dry climates on the other hand it flourished during the winter season, dying out in summer. Rats breed throughout the season in temperate climates, but are retarded in extremes of heat or cold. With the influx of young susceptible rats the epidemic may flare up again. Similarly rat fleas are retarded in the very cold or very hot months. Heat interferes not only with the deposition of eggs, but also prevents the eggs from developing into larvæ. A temperature of 85°F. causes the pestis bacillus to disappear from the intestine of the infected flea rather rapidly. A temperature of 70°F. is optimum for the propagation of an epidemic.

Brooks of the Indian Commission summarized his findings as follows (13):

1. Plague does not maintain itself in epidemic form when the temperature rises above 80° F., accompanied by a saturation deficiency of over 0.30 of an inch.
2. Plague epidemics are rapidly brought to an end in the presence of a high saturation deficiency, even when the mean temperature throughout and after the termination of the epidemic has been considerably below 80° F.
3. Plague epidemics may commence to increase in intensity when the mean temperature is well above 80° F., provided that the saturation deficiency is below 0.30 of an inch.
4. In some districts in India and in certain tropical islands (e.g., Java, Mauritius) where the climatic conditions are at all times of the year favorable to the incidence and spread of plague, the disease may occur indifferently at all seasons.

The Disease in Animals

Plague is primarily a disease of rodents, especially the rat, the ground squirrel, the tarbagan and the veldt rodents of South Africa.

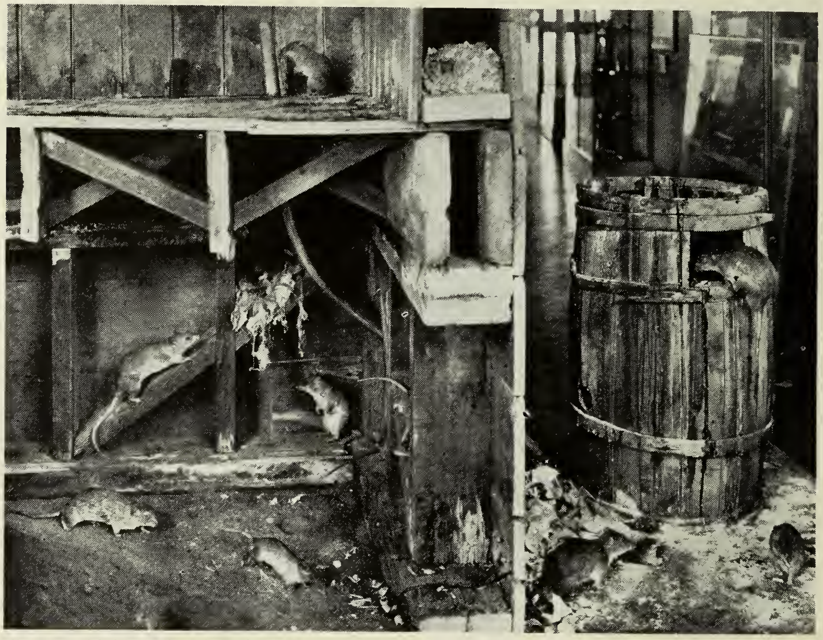
The three rats largely responsible for propagating the disease are the Norway or brown rat *(Rattus norvegicus)*, the Alexandrian or roof rat *(Rattus rattus alexandrinus)* and the English black rat *(Rattus rattus rattus.)* All three of these animals are found over the world, often living together. The Norway rat is the largest, living in the ground under the floors and driving the other rats to the upper stories. He is sometimes found on shipboard; on the ground he is at home, where he flourishes exceedingly, outnumbering the other species in propagation. He is a migrant often traveling many blocks in a night and several miles in a few days. The black rat and roof rat find suitable quarters either on land between the walls or in the upper floors of buildings, or on shipboard between walls and in cargo and merchandise.

Rats do not exhibit marked symptoms of plague till near death. In the natural state they are said to stagger and be easily captured; in cages they may seek a dark corner and try to hide.

The diagnosis of plague in rats is ordinarily based upon macroscopic examination of the gross lesions, which are so characteristic as to leave small doubt in the mind of an experienced observer. In an epizootic, however, about 50 per cent of infected animals will show lesions which are so indefinite that microscopic examination or guinea pig inoculation must be resorted to.

Typical lesions in acute plague are given by Williams as five in number, not all of which will be present at the same time, neither will any one usually appear alone. They are injection, bubo, granular liver, large dark spleen and pleural effusion. Injection of the subcutaneous blood vessels, indicated by a dusky red color of the subcutaneous structures and muscles, occurs in about 75 per cent of infected animals. Buboes in the rat are very suggestive of plague. They may occur in the axilla, neck, groin or pelvis. In India 75 per cent of rats with single buboes had them in the neck, while in San Francisco 75 per cent of such rats had buboes of the groin. The proportion of total rats with buboes varies markedly. In India 80 per cent of animals showed such lesions, in San Francisco 40 per cent, in New Orleans 33 per cent. A granular liver with focal necrosis is present in 60 to 70 per cent of all acute cases. An enlarged spleen is sometimes difficult to determine, as the size of the normal spleen often varies markedly, but different investigators consider this lesion present in 60 to 70 per cent of infected rats. Pleural effusion, often copious in amount, is present in more than 75 per cent of the instances, usually accompanied by one or more of the other lesions. A chronic form may be evidenced by very slight lesions consisting of purulent or caseous foci occurring as abscesses of the liver spleen or mesenteric glands. The plague bacilli may be very scanty in such lesions and often cannot be demonstrated except by guinea pig inoculation. Rats suffering from the chronic form, recovering from the disease, can be diagnosed usually only by the scars present on the various organs. The rôle of such animals in the recurrence of an epidemic is still a matter of conjecture. Plague in rats without lesions was shown by Williams and Kemmerer (3) to exist in New Orleans and Galveston, as well as by other workers in India.

Natural immunity to plague is found in many old rats. The Indian Commission found 59 per cent immune to subcutaneous inoculation of material from the spleen of infected rats. In San Francisco 50 per cent of large rats and 15 per cent of small rats were found resistant to inoculation of virulent material. This immunity is natural and not acquired through previous attacks of the disease.



From exhibit in the American Museum of Natural History, New York

Fig. 22.—Section of tenement house showing conditions that favor the breeding of rats.

The ground squirrels in California, of which *Citellus beecheyi* is the commonest, were first shown to be infected in 1908 by McCoy and Wherry. Probably infection had existed there sometime, however, because in 1903 a blacksmith died of the disease apparently contracted from a ground squirrel, while during the next few years thousands of such squirrels died of an epidemic which probably was plague. Ground squirrels abound in great numbers, making plague endemic in California.

The tarbagan of Siberia *(Arctomys bobac)*, a marmot of which the American woodchuck is a representative, is very susceptible to infection and has been accused of the principal rôle in disseminating plague over the world. Wu Lien-Teh believes that a campaign directed against the tarbagan as well as the rat is necessary for the control of plague.

The veldt rodents of South Africa have recently assumed importance in the spread of plague. Pirie (17) lists sixteen such animals which have been found susceptible to the disease as follows—gerbillie or springhaasrot, namaqua gerbillie, multi-mammate mouse, ground squirrel, striped mouse, long-eared mouse, springhare, hares, white-tailed rat, water rat, karroo rats, fat mouse, dwarf mouse, molerat, yellow mongoose and suricate.

Other rodents are susceptible to plague but do not play an important part in diseminating the disease. In San Francisco a brush rat *(Neotoma)* was found naturally infected by McCoy, while in New Orleans there was trapped a naturally infected field rat *(Hesperomys pulustris)*. Other rats which have been found similarly infected are *Mus musculus* and *Neosocia bandicota* or bandicot rat. The ferret, the skunk, the woodchuck, the weasel, the prairie dog and the gopher are susceptible to the disease and may become infected when exposed (10). Mice are easily infected but are not an important factor in the dissemination of plague. Cats may become artificially infected by eating infected rats, but the disease runs a chronic course usually and the animal is unimportant in the spread of plague. Simson in 1903 reported ingestion plague in calves, chickens, pigeons, dogs, geese, sheep and swine, but other investigators, especially Bannerman and Kapadia and Haffkine and Lawson, obtained opposite results. It is probable that none of these animals are important in the dissemination of infection. Horses have been injected with large doses of virulent plague bacilli with moderate infections resulting, but no instance of natural infection has been recorded in the literature. Several instances are reported of donkeys working in coal mines contracting the disease from coolies infected with pneumonic plague, but these conditions are so extraordinary that the donkey may be overlooked as a source of infection. Camels have been said to be susceptible to natural infection and a possible vehicle of spreading infection from place to place along caravan routes. This possibility is denied by Castellani and others. Opinion as to the possibility of infection of bats is divided. Birds that run on the ground are quite generally resistant, however. The booby owl, resistant to plague infection itself, often shares the quarters with the California ground squirrel and may be responsible for the dissemination of infected fleas.

FLEAS

Fleas were experimentally shown by Simond in 1897 to be a possible agent for the dissemination of bubonic plague, either from animal to animal, animal to man, or man to man. The last Indian Commission fully corroborated this view. Infection may at times take place by regurgitation at the time of flea bites, or the organisms may be passed out with the dejecta and rubbed into the skin. The blood stream of an infected rat may contain as many as 100,000,000 organisms per cubic centimeter. At one feeding a rat flea may take in 5,000 organisms, which then develop in the digestive tract of the flea.

Following are fleas of more or less importance in the regard to bubonic plague:

European rat flea—*Ceratophyllus fasciatus*
Indian rat flea—*Xenopsylla cheopis*
Mouse flea—*Ctenopsylla musculli*
Human flea—*Pulex irritans*
Dog flea—*Ctenocephalus canis*
Cat flea—*Ctenocepalus felis*
Squirrel flea—*Ctenocephalus acutus*
Squirrel flea—*Hoplopsyllus anomalus*
Tarbagan flea—*Ceratophyllus silvantieri*

The rat flea of Europe and North America, *Ceratophyllus fasciatus,* will feed upon man in the absence of rodents. They may hibernate without food for a month to forty-seven days, or even receive food regularly for the same length of time and remain infective. The Indian rat flea, *Xenopsylla cheopis,* is the responsible agent, not only in India, but in other parts of the world likewise for much of the spread of plague. In New Orleans in 1916 the fleas on 1,268 rats numbered as follows: *Xenopsylla cheopis* 3,256, *Ceratophyllus fasciatus* 88, *Leptopsylla musculi* 1,263, *Ctenocephalus canis* 125, and *Pulex irritans* 47. These proportions varied with the seasons and the years, but *Xenopsylla cheopis* always predominated (7). The human flea, *Pulex irritans,* is rare in most parts of the United States. It is capable of transferring plague experimentally as are the cat flea, *Ctenocephalus* *canis,* and probably others, but it is doubtful if they are of practical importance.

Cragg (15) has shown that in those parts of India where plague does not exist the predominating rat flea is not *Xenopsylla cheopis,* but other species. In Colombo, which enjoys a relative immunity from plague, the common rat flea is *Xenopsylla astia.* In the United States a survey by the Public Health Service showed that in New Orleans, where plague actually existed, *Xenopsylla cheopis* was the predominating flea, while in New York and Boston this pest appeared for only a few months of the year.

The total number of fleas per rat varies with the climate, the seasons and other factors. In Belgaum, India during July, August and September, which is the epidemic season, 18 fleas per rat were found while in the colder, non-epidemic season, only 4 or 5 were present. In the United States in New Orleans in 1922 the average number of fleas in the winter time was about three while in the summer it mounted to nearly five. In New York City the average number of fleas on 4,756 rats for a two year period was 0.93, with the largest number from any single rat 38 (7).

The Disease in Man

Plague in man may manifest itself in various forms, resulting in such designations as bubonic plague, pneumonic plague, septicemia plague and ambulatory plague.

Bubonic plague is by far the most common type, accounting for about 75 per cent of cases. It takes its name from the fact that the lymph glands draining the area upon which the infection occurs become enlarged (buboes). The disease is accompanied by fever, depression, and great prostration. The enlarged lymph glands are extremely tender and painful. The glands of the groin are most commonly affected because the flea-bites more often occur on the legs; axillary and cervical glands follow in order. The incubation period varies from two to ten days but usually averages four to six days. The onset is sudden.

Bubonic plague in man in nature is practically always caused by the bite of an infected flea. The disease therefore is limited to those climates and countries where the flea of the rat (or of other rodents) have ready access to man. Wherever a fairly high degree of sanitation is carried out, excluding the possibility of contact of rats and man, there is small danger of human infection. The disease is further self-limited by climates and seasons.

Robertson has shown that in countries where the mean mid-winter temperature is 45°F. or below the disease is only occasional and self-limited. The seasonal prevalence follows the breeding of fleas, cases occuring in the temperate climate of the United States mostly during the summer and fall, while in the tropical climate of Bombay they occur during the winter and early spring. Bubonic plague in man is therefore a disease of tropical or sub-tropical countries, flourishing best during moderately warm dry weather. In colder climates epidemics can occur for only short periods of time.

Pneumonic plague is an infection of the lungs with the plague bacillus, resembling pneumonia. Transfer of the infection from person to person takes place by way of the nose and mouth from an existing case of the disease, as with other respiratory infections. Neither the rat nor the flea enter into the epidemiology of the trouble except to initiate it. Persons with bubonic plague may develop an infection of the lungs after which they are capable of infecting other persons. Sometimes infection from ground squirrels develops into the pneumonic type, possibly because of a special strain of organism that has predilection for the lungs. The disease is rapid in its course, with an incubation period of one to three days, followed by almost certain death two to four days later. The sputum of such patients is copious in amount, containing enormous numbers of plague bacilli, making the transfer of infection easy. Norman White believes that pneumonic plague is due to a secondary invader.

Pneumonic plague is a disease of cold climates and winter seasons. The first large epidemic of this type of plague since the advent of bacteriology took place in Manchuria in 1910-11. Most of our knowledge concerning it was gleaned at that time. Sixty thousand persons perished during that epidemic, with another similar one ten years later carrying off 9,000 more people. In the United States two small epidemics of pneumonic plague have originated in California from ground squirrels, one in 1919 and a larger one in 1924, but both quickly subsided.

Septicemia plague is a general acute infection of the blood stream with the plague bacillus. It usually is of short duration, eighteen hours to three days, killing the patients before buboes have time to appear.

Ambulatory plague is a very mild form of the disease. Since buboes develop it can be classed under the head of bubonic plague.

Prevention

The control of bubonic plague lies largely with the control of rats. Other rodents may become infected but because of lesser association with man, they are of small importance in spreading contagion. Exception to this statement may be taken with the ground squirrel in California and with the tarbagan in Manchuria where these animals act as foci for new outbreaks of the disease. The number of rats in the United States is impossible to estimate, but they far exceed the human population. The cost of supporting them was estimated by Creel in 1913 as $167,000,000 annually.

The destruction of rats may be accomplished by several methods. Traps are serviceable but care must be used in preparing the bait, to eliminate the "man smell," and judgment used in setting the traps, for rats are wary creatures. Chemical poisons consist of barium carbonate, strychnine, arsenic or phosphorus. Barium carbonate is very efficient when mixed with four parts of flour into dough. Arsenic is prepared in a similar manner. Strychnine has no advantage over the other poisons and has the disadvantage of accidental human poisoning at times. Phosphorous is sometimes used but is a fire hazard. Bacterial rat viruses such as *Salmonella typhimurium* or *Salmonella enteriditis* are not recommended by most authorities. Epidemics among rats due to such viruses are limited in extent, the viruses rapidly lose their virulence, and the danger to man has been demonstrated on numerous occasions. Shooting is serviceable to reduce the number of rats and to discourage others. Well trained dogs, such as fox terriers, are very useful in combating the rat population; cats are usually too indolent or are not capable of overcoming large rats. It is most important to keep rats from garbage and otherwise restrict their access to food. Fumigation of buildings or ships destroys enormous numbers of rats. All ships sailing from plague-infected ports must be fumigated before entering a port of the United States, hydrocyanic gas usually being used. For stables, cellars and buildings, sulphur dioxide is serviceable since it does not have the danger to human life which hydrogen cyanide does.

Rat-proofing of buildings is of great importance. This may be accomplished by reinforcing the lower wooden parts with metal to prevent gnawing through, screening windows, doors and other exits to prevent entry, and guarding openings around water, sewer and gas pipes. Ships should carry rat guards on the hawsers to prevent the escape of rats from vessels as well as boarding from wharves.

Human cases of plague must be given particular attention. Bubonic cases should be isolated in vermin-free surroundings. Infected fleas may be carried from place to place by healthy individuals. Pneumonic plague requires a different type of campaign, stress being laid upon the respiratory nature of the disease. Face masks have been found serviceable in preventing infection.

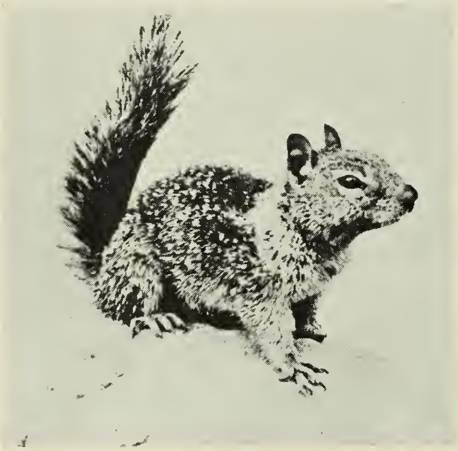


FIG. 23.—Ground squirrel (Citellus beecheyi), reservoir of plague in the United States.

Vaccines of various kinds have been advocated for the control of bubonic plague. The most popular is that of Haffkine, consisting of killed organisms which are administered subcutaneously, given in two injections ten days apart. Other workers, especially Kolle and Strong, used living attenuated organisms. Various other recomendations have been made, none of which have proved entirely satisfactory. The Indian Plague Commission reported that vaccination diminishes the incidence of plague but does not furnish absolute protection. Teague corroborated this evidence, finding the percentage of mortality greatly lessened in vaccinated persons.

Beals (6) in India reported among 6,000 unvaccinated in Wai in 1916-17 there were 275 cases and 167 deaths, while among 4,378 protected with Haffkine's vaccine there were only 39 cases and 10 deaths. The next year among 6,000 uninoculated there were 76 cases and 47 deaths, while among the vaccinated there were 17 cases with 2 deaths. McCoy and Chapin however, assert that vaccination has never been known to control a plague epidemic. This measure must not be allowed to interfere with other tried and proved methods of control. Pneumonic plague is not influenced by vaccine.

Plague serum from immune horses has been recommended by Yersin but its protective powers are transient and its curative powers feeble. It has not attained a prominent place in plague control.

Ground squirrels may be destroyed by methods used by the United States Public Health Service in California. The most effective procedure is the vaporizing of carbon disulphide into the holes. Poisoned bait is also effective. Traps are not successful because of the wariness of the animal. The natural enemies of the squirrel—the coyote, wolf, badger, skunk, snake, hawk and mountain lion—kill many of them and should be encouraged.

Items of Note

1. Plague is primarily a disease of rodents.
2. Bubonic plague is a disease of tropical or semi-tropical climates.
3. Pneumonic plague is a disease of cold and temperate climates.
4. The control of rats is sufficient to control bubonic plague in man.
5. Pneumonic plague lends itself to no practical control methods
6. In the United States the ground squirrels of California are naturally infected, tending to perpetuate the disease.

BIBLIOGRAPHY

1. Wu Lien-Teh
   Japan Medical World, 1924, *4,* 1-8.
2. Greenberg, D.
   Sci. Monthly, 1917, 554-566.
3. Williams, C. L., and Kemmerer, T. W.
   Public Health Reports, 1923, *38,* 1873-1881.
4. Williams, C. L.
   Am. Jour. Public Health, 1920, *10,* 851-864.
5. Rucker, W. C.
   Jour. Am. Med. Asso., 1915, *65,* 1767-1768.
6. Beals, L. H.
   Jour. Am. Med. Asso., 1920, *75,* 955-956.
7. Fox, C., and Sullivan, E. C.
   Public Health Reports, 1925, *40,* 1909-1934.
8. Wherry, W. B., Walker, Agnes, and Howell, E. H.
   Jour. Am. Med. Asso., 1908, *50,* 1165-1167.
9. Strong, Richard P.
   Jour. Med. Res., 1908, *18,* 325-346.
10. McCoy, G. W.
   Jour. Inf. Dis., 1911, *8,* 42-46.

11. Kitasato, S.
Lancet, 1894, *2,* 428.

12. Yersin
Ann. de l' Inst. Past., 1894, *8,* 662.

13. Brooks, R. S.
Tenth Report Plague Investigation in India, Plague Suppl. V., 1917, 881-889.

14. McCoy, G. W., and Wherry, W. B.
Jour. Inf. Dis., 1909, *6,* 670.

15. Cragg, F. W.
Indian Jour. Med. Res., 1921, *9,* 374-397.

16. Lloyd, B.J.
Jour. Am. Med. Asso., 1925, *85,* 729-733.

17. Pirie, J. H. Harvey
Publications of South African Inst. Med. Res., 1927, *3,* 109.

CHAPTER XV

TULAREMIA

"Tularemia is an infectious disease caused by *Bacterium tularense*. Primarily it occurs in nature as a fatal bacteremia of wild rodents, especially rabbits and hares. Secondarily it is a disease of man, transmitted from rodents to man by the bite of an infected blood-sucking fly or tick, or by contamination of his hands or his conjunctival sac with portions of the internal organs or with the body fluids of infected rodents, flies or ticks," (Francis).

Since 1910 the disease now called tularemia has been recognized under various names according to the circumstances in which it was encountered. Thus McCoy designated it as "a plague-like disease of rodents"; in Utah it was known as "deer-fly fever" and in Washington, D.C. as "rabbit fever"; in Idaho Dr. Lamb referred to it as "the glandular type of tick fever," in Japan it has been reported as "Ohara's disease."

HISTORY

A spectacular glamour attaches itself to the discovery of tularemia. It is a new disease, being recognized as such only since 1921. Its identity and complete description were worked out entirely by the collaboration of American investigators, the only such incident in history. As the problem of yellow fever was solved by members of the United States Army, so tularemia was the result of observations and research made by members of the United States Public Health Service. Also as yellow fever took toll of that intrepid band of soldiers in Cuba, so has tularemia laid low those who sought to conquer it. It is a curious fact that all six members of the force connected with tularemia investigations in 1919 to 1921 contracted the disease; even the most elaborate precautions failed to prevent infection.

In 1911 (1) McCoy working in Tulare County, California, described a "plague-like disease of rodents" in California ground squirrels. The next year he and Chapin isolated the causative agent of the disease and named it *Bacterium tularense* (2). Both Chapin and a laboratory assistant had gone through an obscure attack of fever in the course of their work and it was found

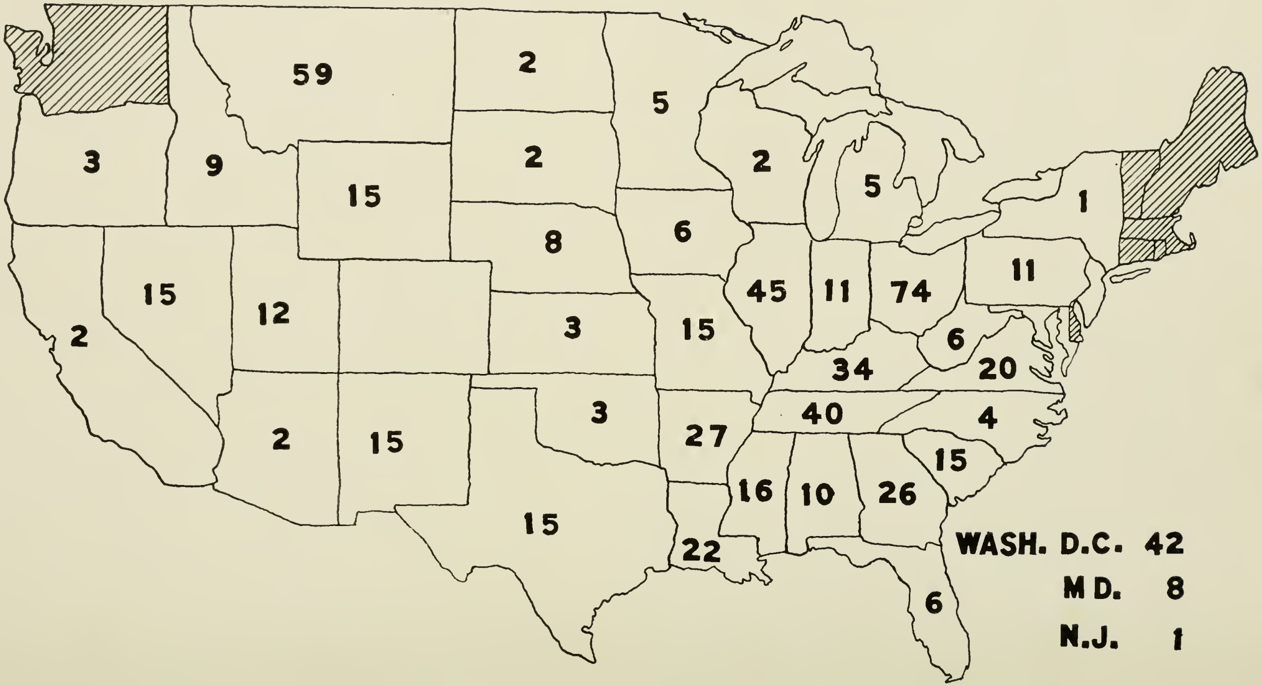

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Fig. 24.—Cases of tularemia in man reported in the United States up to 1928. Many cases probably occurred of which there was no record. The State of Washington, the New England States and Delaware were apparently free of the disease.

afterwards that their blood serum agglutinated this organism. In the years that followed there were reported isolated instances of recovery of *Bacterium tularense* from human cases.

In Cincinnati three cases occurred among the patients of ophthalmic physicians. In 1914 Vail described an eye infection from which Wherry and Lamb (3) isolated *Bacterium tularense,* this being the first human case of tularemia on record to be diagnosed bacteriologically. In 1915 Sattler (4) and in 1917 Lamb (5) described similar cases of conjunctivitis.

In 1920 Francis isolated *Bacterium tularense* from seventeen jackrabbits and seven human cases of "deer-fly fever." This disease had been known in Utah since the publication of a paper by Pearse (11) in 1911 and was so named because it was popularly supposed that infection was due to the bite of the blood-sucking fly, *Chrysops discalis,* commonly found on horses. Francis proved that the deer-fly actually did transmit *Bacterium talarense.* He named the disease tularemia because of the presence of the tularense organism in the blood (analagous to leukemia and bacteremia).

Geographical Prevalence

Tularemia has been found in most sections of the United States, in Japan (6) and in Russia. It has never been discovered elsewhere however. Although the disease was not established as a separate entity until 1921, by consulting the records of symptoms and laboratory tests it has been possible to show that it existed in certain localities before that time.

The extent of the disease in animals is not known exactly, and whether it exists in the territory beyond which human cases have been found is problematical. Without doubt the prevalence within the area mentioned is more serious than is ordinarily realized. Recently I was informed that the rabbits in a certain section of Illinois were rapidly dying from tuberculosis. They were so weak that they could be caught easily with the hands; the liver showed many small lesions resembling tubercles. Since bovine tuberculosis was not very prevalent in that locality and since rabbits in the wild state have not been known to suffer from spontaneous tuberculous infection, the disease in question was without doubt tularemia. It has been found since that time in rabbits in that section of Illinois.

Seasonal Prevalence

Tularemia has no seasonal prevalence except as it is influenced by insects or by contact with infected animals. Laboratory infections may occur at any time. Human cases are most prevalent in summer time in the western states where ticks and deer flies are the cause of infection (*Dermacentor andersoni,* March to August; *Chrysops discalis,* June to September). Human cases, due to infection from dissecting jackrabbits, are likewise most prevalent during April to October because these are the months of greatest destruction of these pests. East of the Mississippi River cottontail rabbits are responsible for most human cases and since they are generally protected by law except during a few winter months, the seasonal prevalence is apt to be November to January, when the hunting season is on.

Table 36

STATES IN WHICH TULERAMIA HAS BEEN FOUND TO EXIST

With date of first known cases

California	1904
Arizona	1907
Ohio	1908
Utah	1908
Missouri	1909
Idaho	1914
Illinois	1914
Indiana	1914
Wyoming	1915
Virginia	1920
North Carolina	1921
Washington, D.C.	1921
Tennessee	1922
Louisiana	1923
Montana	1923
Mississippi	1924
New Mexico	1924
West Virginia	1924
Arkansas	1925
Georgia	1925
Iowa	1925
Kansas	1925
Kentucky	1925
Maryland	1925
Oregon	1925
Pennsylvania	1925
South Carolina	1925
Texas	1925
Alabama	1926
Colorado	1926
Florida	1926
Michigan	1926
Minnesota	1926
Nebraska	1926
Nevada	1926
North Dakota	1927
Oklahoma	1927
South Dakota	1927
New Jersey	1928
New York State	1928
Wisconsin	1928

Animals Susceptible

Francis noted the degrees of susceptibility in animals as follows:

High suscepibility—man, monkey, ground squirrel, rabbit, guinea pig, mouse, woodchuck, opossum, young coyote, pocket gopher, porcupine, chipmunk and grouse.

Slight susceptibility—rats, cats, sheep and goats.

No susceptibility—horses, cattle, hogs, dogs, pigeons, chickens, turkey and fox.

Infectious Agent

Bacterium tularense (designated as *Pasteurella tularensis* in Bergey's Manual of Determinative Bacteriology) is a small rod 0.2 by 0.3 to 0.7 microns, occuring singly. It is non-motile, Gram negative and non-spore bearing. It is extremely pleomorphic yielding bacillary, coccoidal and bipolar forms. Growth on ordinary culture media does not take place, but blood agar, dextrose blood agar and dextrose serum agar allows some development. The addition of fresh rabbit spleen to the medium favors growth, but the best media are coagulated egg yolk or blood-dextrose-cystine-agar.

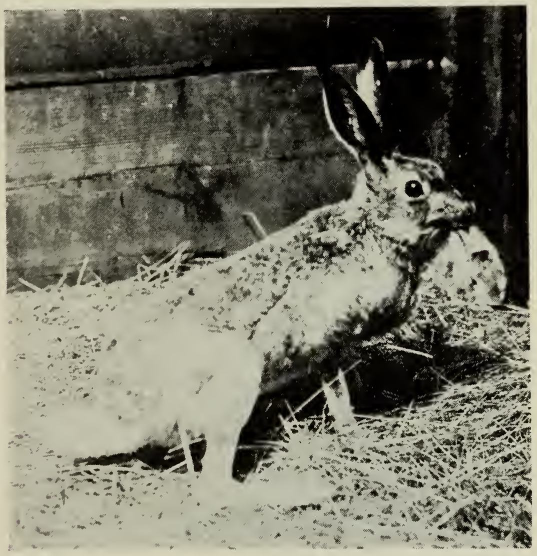
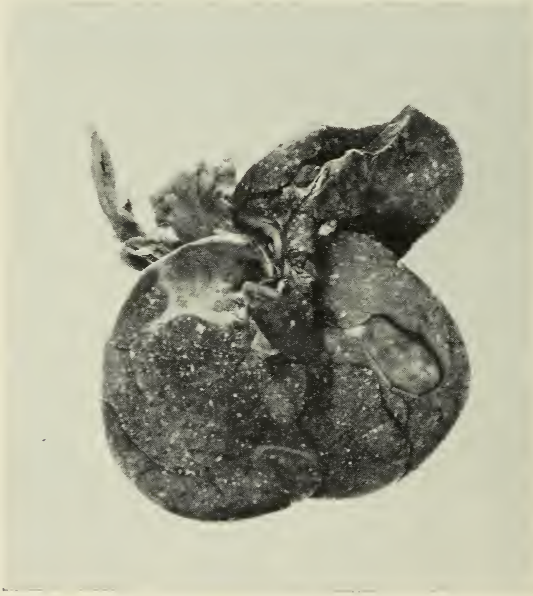


Fig. 25.—Jack rabbit, common host and transmitter of tularemia west of the Mississippi river.

The organism is easily killed by heat in ten minutes at 56°C. to 58°C. Cooking therefore renders infected tissue harmless. Chemical disinfectants destroy the organism easily—tricresol 1.0 per cent in two minutes and formalin 0.1 per cent in 24 hours. Glycerin pure and undilated, is suitable for preserving spleens for laboratory inoculations, the organism remaining alive many days when the spleen of an affected rabbit or guinea pig is thus treated.

The Disease in Animals

Tularemia is primarily a disease of wild rodents, especially rabbits and hares. It has never been found in tame rabbits in hutches, though such animals are susceptible to infection, if it is carried to them. Cottontail rabbits, jackrabbits and snowshoe rabbits form the great reservoir of infection in the United States; in Japan likewise rabbits have been found infected. Ground squirrels were found infected by McCoy, wild rats by Dieter and Rhoades (7), wild mice by Perry (8), sheep by Parker and Dade, ruffed grouse by Green and Wade, opossums by Mease and Dunedin and water rats by the Russian workers.



Courtesy of Dr. Edward Francis, U.S.P.H.S.

Fig. 26.—Tularemia. Rabbit's liver, showing small necrotic foci of necrosis.

Transmission of tularemia from animal to animal is probably carried on by means of blood sucking insects, especially lice, fleas, and ticks. Rabbit lice are active the year around; wood ticks are a menace during the spring and summer in not only carrying the infection from rabbit to rabbit directly but also in transmitting it to its offspring through the egg. The deer fly, while usually found around horses, may bite rabbits, mechanically transmitting the infection from animal to animal through its infected mouth parts. The mouse louse is probably the cause of the spread of the disease in mice.

While the gross lesions vary somewhat in different animals they resemble very closely bubonic plague in the guinea pig. Guinea pigs readily contract the disease if a minute amount of the virus is placed upon the shaved skin, death occurring in five to seven days. The site of the inoculation is necrotic; the contiguous lymph glands are hemorrhagic or caseous while a secondary chain of lymph glands may be caseous; the liver is covered with small yellowish gray necrotic foci; the spleen is enlarged and thickly studded with miliary necrotic granules, so thick that large areas of the spleen are at times necrotic.

A chronic form of the disease has been reported in guinea pigs inoculated with attenuated cultures. There is marked enlargement of lymph nodes and other organs which, with necrotic foci, suggests tuberculosis.

Francis has never found evidence of immunity in animals except in one rabbit which survived a severe attack and was thereafter resistant to an injection of one million fatal doses of culture.

The Disease in Man

Tularemia is secondarily a disease of man transmitted from animals by means of blood sucking insects, by direct contact with infected animals or sometimes by the bite of an animal which has recently been feeding on an infected animal.

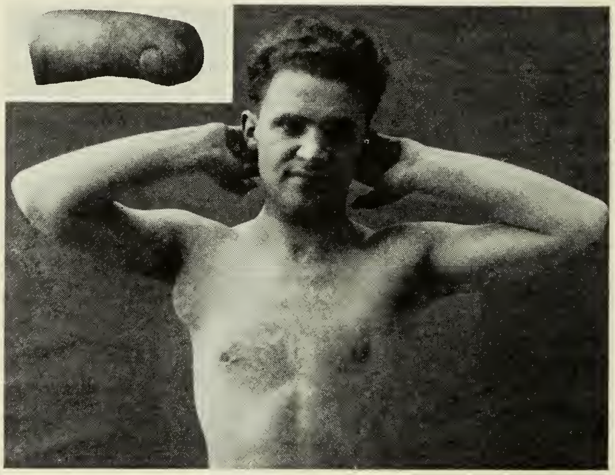
The following arachnids and insects have been shown capable of transmitting the disease:

Woodtick, *Dermacentor andersoni.*
Tick (species undetermined, probably *Dermacentor variabilis*).
Rabbit louse, *Haemodipsus ventricosus.*
Deer fly, *Chrysops discalis.*
Bed bug, *Cimex lectularius.*

Mouse louse, *Polyplax serratus.*
Squirrel flea, *Ceratophyllus acutus.*
Stable fly, *Stomoxys calcitrans.*

All but the rabbit louse and mouse louse bite man as well as animals. The deer fly and the woodtick however, are the worst offenders. The deer fly, while commonly found around horses and cattle, may also feed on rabbits, transmitting the infection directly to man by mechanical means.

The habitat of the deer fly is Utah and surrounding states. The woodtick feeds on both rabbits and man and thus may transmit the infection directly through its contaminated mouth parts. More serious, however, is the transmission of the infection to the second generation of the woodtick, through the egg. Ticks carry their infection over winter and throughout their lives, thus forming a permanent reservoir of tularemia. One species of woodtick is found in Montana and surrounding states while another species is found in southern United States.



Dr. H. Hertzberg, Washington, D.C.

FIG. 27.—Tularemia. Ulcer of right finger after dressing market rabbits. Right axillary abscess. 49 days after onset.

Although the outbreak of tularemia in Russia was due to direct contact with rats, so far as is known there is no record of infection to man from rats or from mice or ground squirrels through insects.

Infection through abrasions is the chief mode of spread to man. Since *Bacterium tularense* is found in the blood, urine and salivary secretions of infected rabbits, it is to be expected that any abrasions on the hands of those skinning or handling such animals would expose them to infection. Numerous instances are on record of persons scratching or pricking themselves while skinning rabbits and later developing the disease—the hunter, the market man, the housewife, or the cook. Eye infections are not uncommon, caused by rubbing the eye with a finger contaminated while handling infected animals or insects. Especially is this true in Montana where a farmer pulls infected ticks from his horses and cattle, then touches his eye. Numerous laboratory infections have occurred through unknown channels by contact with infected guinea pigs, rabbits or white mice or handling infected living ticks. Every precaution known to medical science has been taken to prevent infection in the laboratory, to no avail. It is assumed that the transmission is by means of the respiratory tract, possibly through inhalation of contaminated dust, or, as Francis believes more likely, directly through the skin of the hands.

A few instances are on record of infection caused by the bite of animals. Francis mentions three—a coyote, a ground squirrel and a hog (9). In each instance it was presumed that the mouth parts of the animals had become contaminated by infected rabbits which they had eaten, for such animals were found nearby readily accessible.

No instances are on record of tularemia being passed from person to person except in one case where a woman who pricked her thumb while dressing the lesion on the neck of her fly-bitten son is believed to have become infected. No epidemics of tularemia may be expected to break out in our cities therefore.

The prevalence of tularemia in man in the United States is comparatively small, but possibly larger than is suspected. Francis has collected records of several hundred cases over the country occurring in a period of five years, and probably there were many more than this. Stewart (10) in Kansas made routine agglutination tests for tularemia on blood specimens submitted for Wassermann tests. Out of 512 such sera there were 8, or 1.5 per cent, showing positive reactions. Histories of the patients, obtained subsequently, indicated that those giving positive tests had at one time had tularemia.

There are four clinical types of the disease reported by Francis in studying 679 cases.

1. Ulceroglandular, the primary lesion being a papule, later an ulcer of the skin and accompanied by enlargement of the regional lymph glands.

2. Oculoglandular, the primary lesion being a conjunctivitis and accompanied by enlargement of the regional lymph glands. Fulminant cases, running a rapid course, with death, have been noted in the oculoglandular type.

3. Glandular, without primary lesion, but with enlargement of the regional lymph glands.

4. Typhoid, without primary lesion and without glandular enlargements.

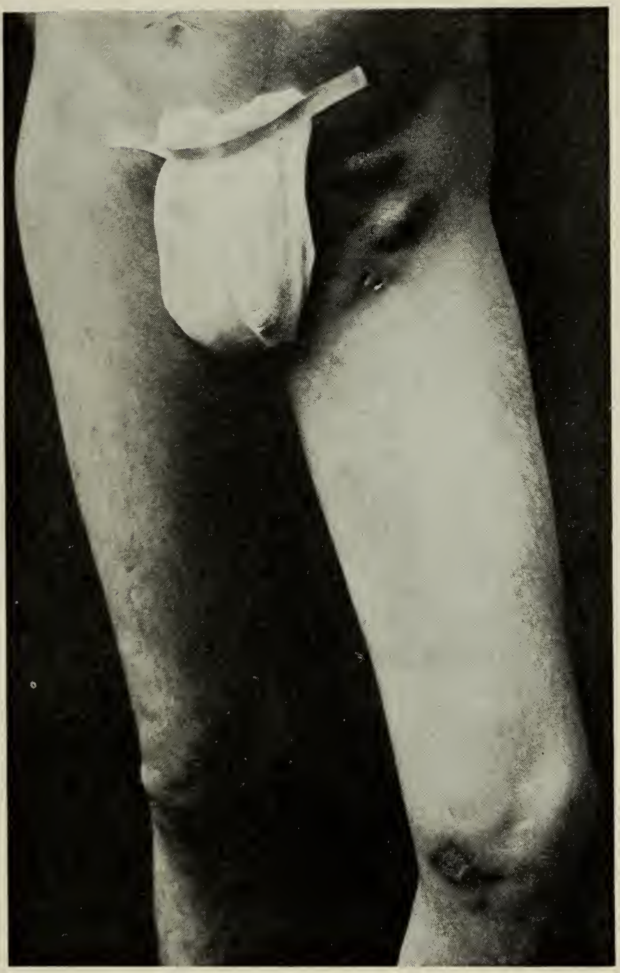
Sources of infection were studied by Francis in 540 cases as follows (9):

Ulceroglandular type, 455 cases.
23 were fly-bitten *(Chrysops discalis)*.
33 were tick-bitten *(Dermacentor andersoni)*.
21 were tick-bitten *(Dermacentor variabilis)*.
6 were associated with sheep and ticks.
4 were bitten by insects (species?).
100 were market men who had dressed rabbits.
25 had dressed rabbits bought in the market.
98 had dressed rabbits which they had shot.
34 had dissected jack rabbits.
131 had dressed rabbits the source of which was not stated.
1 had killed and skinned a woodchuck.
1 was bitten by a coyote.
1 was bitten by a ground squirrel.
1 was bitten by a hog.
1 was scratched by a cat.

Oculoglandular type, 32 cases.
21 had skinned or dressed wild rabbits.
8 had crushed ticks with their fingers.
1 had crushed a fly with his fingers.
2 source of infection was uncertain.

Glandular type, 25 cases.
24 had dressed rabbits.
1 was an experimental human subject.

Typhoid type.
20 had performed autopsies on laboratory animals or handled ticks.
6 had dressed wild rabbits.
1 was tick-bitten.
1 was probably tick-bitten.



Medical Clinics of North America

FIG. 28.—Tularemia. Tick bite left knee. Left inguinal gland. 70 days after onset.

How the infection gained entrance to the body in laboratory workers is unknown. Whether it penetrated the skin of the hands without leaving a local lesion or whether, after contamination of the fingers, the infection was conveyed to the conjunctival sac or to the mouth and swallowed, or whether it was conveyed as a droplet infection remains undetermined. There is little evidence to support any view.

Tularemia is often mistaken for septic infection, tuberculosis, influenza, typhoid fever, undulant fever, or sporotrichosis. The incubation period varies from 24 hours to 9 days, being in most instances about 3 days. The onset is sudden often occurring while the patient is at work. Convalescence is slow, many times being from three to six months. Relapses sometimes occur lasting several days. The death rate is about 4 per cent, only 24 cases dying out of 679 studied by Francis. Diagnosis may be made certain by laboratory agglutination tests and by the isolation of the organism. Agglutination tests are not positive during the first week of illness but begin to show in the second week and persist for many years after recovery. Animal inoculation of suspected material is attended by grave danger to the laboratory worker, as practically every such person has sooner or later contracted the disease.

There is no treatment for tularemia other than symptomatic. No curative or preventive serum or vaccine has been perfected. The relative importance of tularemia for man is small. It will never exist in epidemic form in the human population nor will it be passed from man to man under ordinary circumstances. It usually occurs as a rural disease after direct contact with wild rabbits or infected ticks. The danger of infection however, is very real to those persons who come into contact with such animals or insects.

PREVENTION

There is no known method of preventing the spread of infection among animals. Man can protect himself by avoiding contact with infected animals. Laboratory workers should wear rubber gloves and take all possible precautions known to medical science. Hunters, market men and cooks should wear rubber gloves while skinning and dressing rabbits.

Thorough cooking destroys infection, thus rendering a rabbit safe for eating purposes, even though it may have harbored the disease.

There is no known preventive serum or vaccine for man or animal.

ITEMS OF NOTE

Francis cites the following unique features of tularemia which deserve special mention:

1. The certainty of infection of laboratory workers.
2. The persistence of agglutinins in the blood of long recovered cases.
3. The cross-agglutination of abortus and melitensis.
4. The granulomatous character of the lesions in man as contrasted with the lesions in animals.
5. The cystine requirement of the organism in culture medium.
6. Its pleomorphism.
7. Its penetration of the unbroken skin.
8. Its invasion of fixed tissue cells—the hepatic cells of a mouse and the intestinal epithelium of tick and bed bug.
9. Its hereditary transmission through the egg of the tick to the next generation of ticks.
10. The great variety of insect hosts.
11. The great variety of animal hosts.

BIBLIOGRAPHY

1. McCoy, G. W.
   U. S. Public Health Service, Public Health Bull. 43, 1911.
2. McCoy, G. W., and Chapin, C. W.
   U. S. Public Health Service, Public Health Bull. 53, 1912.
3. Wherry, W. B., and Lamb, B. H.
   Jour. Inf. Dis., 1914, *15*, 331-340.
4. Sattler, Robert
   Arch. Ophthal., 1915, *44*, 265.
5. Lamb, F. W.
   Ophth. Rec., 1917, *26*, 221-226.
6. Francis, E., and Moore, D.
   Jour. Am. Med. Asso., 1926, *86*, 1329.
7. Dieter, L. V., and Rhodes, Bernice
   Jour. Inf. Dis., 1926, *38*, 541.
8. Perry, J. C.
   Public Health Rep., 1928, *43*, 260-263.
9. Francis, E.
   Jour. Am. Med. Asso., 1928, *91*, 1155-1161.
10. Stewart, A. V., and Moss, Mildred
    Jour. Arkansas Med. Soc., 1927, *24*, 64.
11. Pearse, R. A.
    Northwest Medicine, March, 1911.

CHAPTER XVI

SPIROCHETAL JAUNDICE

Spriochetal jaundice is an acute infection caused by the spirochete *Leptospira icterohemorrhagiae.* Primarily it is an affection of rats, secondarily a disease of man. Other names by which the infection has been designated are Weil's disease, leptospirosis icterohemorrhagiae, spirochetosis icterohemorrhagiae, acute hemorrhagic jaundice, epidemic jaundice and infectious jaundice. There are many cases of infectious jaundice however, which are not of spirochetal origin.

Infectious jaundice has been known to occur for many years among soldiers, sewer workers and ditch diggers. In the War of 1812 there were numerous cases among the soldiers, while in the American Civil War there occurred among the Northern troops more than 71,000 cases. The mild form of the disease together with the lack of fatalities would indicate that these outbreaks were not of spirochetal origin but rather an epidemic catarrhal jaundice of unknown etiology. Blumer (1) has reported many such outbreaks in North America.

Under the heading infectious jaundice in the past a large group of conditions have been included, from which from time to time have been separated such jaundices as those connected with malaria, yellow fever, typhoid fever, relapsing fever and the like. Spriochetal jaundice is the most recent to be separated as a distinct entity.

In 1914 Inada and Ido in Japan transmitted to guinea pigs a disease similar to Weil's disease in Europe. They discovered in human specimens as well as in the blood and organs of the guinea pigs an organism which they named *Spriocheta icterohemorrhagiae* (2). It was not long before these results were confirmed in the armies of both the Allies and the Central Powers in the World War. The German investigators Hübener and Reiter (3) gave to the name of the organism isolated by them *Spriocheta nodosa.* Noguchi (4) after making a comparative study of the spirochetes isolated in America, Europe and Japan, proposed a new genus *Leptospira.*

Leptospira icterohemorrhagiae has been found in various parts of the world. In Japan Miyajima isolated the spirochetes from the kidneys of field mice while Ido, Hoki, Ito, and Wani (6) demonstrated them in the organs of house rats, *Mus norvegicus,* as well as from persons suffering with Weil's disease. They have been found in the kidneys of wild rats in Belgium by Stokes, Ryle and Tytler (7); in England by Coles (8), by Foulerton (9), and by Gulland and Buchanan (10); in Guayaquil by Noguchi (11); and in the United States by Noguchi in New York (4), Jobling and Eggstein in Nashville (13), Wadsworth in Albany (14), Robinson in Baltimore (15), Neil in Washington (16), Otteraaen in Chicago (17) and Walch in Baltimore (34).

THE ETIOLOGICAL AGENT

Leptospira icterohemorrhagiae is a spiral organism with irregular wavy undulations with one or both ends sharply hooked when seen in free space. In semi-solid media the organisms appear bent, waved or serpentine. They are cylindrical in shape about 0.25 to 0.3 microns by 7 to 14 microns with sometimes long forms 30 to 40 microns. The ends are usually drawn out into fine points, although when resting they may appear blunt. The motility is very rapid, with a cork-screw or serpentine movement along the whole length of the organism. There is no flagellum, however.

The spirochetes do not stain readily with aniline dyes, but with Giemsa stain they can be easily demonstrated. The dark field illumination of the microscope is the most satisfactory method to detect their presence in suspected tissue, even better than animal inoculation. Ido found 32 per cent of specimens positive by dark field and 7 per cent by animal inoculation. Robin's figures were 7 per cent positive with dark field and 4 per cent by animal inoculation.

The spirochetes have been cultivated on a variety of media, the blood serum of man or animal being necessary. Bile salts completely dissolve the organisms, but to saponin they are completely resistant.

Noguchi (12) found that the spirochetes were unable to grow in urine and could not even survive for 24 hours unless the acidity of the urine was neutralized. If suitable ingredients for culture were added to neutralized urine, the organisms might grow for 10 days but no longer. In feces the spirochetes die within 24 hours.

In polluted water, sewage and soil they will not remain alive more than three days. Many of the bacteria found in soil, feces, water and sewage are antagonistic to the growth of the spirochetes.

Pagniez (32) and his associates added 5 per cent of a 40 per cent formaldehyde to the urine to prevent fermentation and acidity. Spirochetes could be demonstrated by this means in urine 22 days old. The Fontana stain was found most suitable.

Leptospira icterohemorrhagiae is closely related to *Leptospira icteroides,* found in cases of yellow fever by Noguchi (18). He differentiated the two organisms by means of serological and immunological reactions. Theiler and Sellards (19) however, could find no difference between them by these reactions. They conclude that either *Leptospira icteroides* has nothing to do with yellow fever or that spirochetal jaundice and yellow fever are one and the same disease. Further research is necessary before the exact status of the two organisms is determined.

Animal Infections

Rats are apparently not at all inconvenienced by *Leptospira icterohemorrhagiae.* The spirochetes are found in large numbers in the kidneys and are excreted in the urine. They are present in the liver and other organs and may be excreted in the feces.

Studies in various parts of the world have indicated that about 10 per cent of rats harbor the organism except in the vicinity where human infection exists, when the rate goes as high as 40 per cent.

Probably all breeds of rats are susceptible to infection. Robinson in Baltimore worked with *Mus norvegicus.* Ido and his associates in Japan found in 100 *Mus decumanus* 40.2 per cent infection and in 24 *Mus alexandrinus* only 0.8 per cent. These differences may have been the result of an actual difference in resistance to infection, or they may have been the result of differences in opportunity for infection, the age of the rats or some other factor.

Old rats with scars are most likely to show infection, while young rats rarely show it. The manner of infection from rat to rat is obscure. They may infect each other through bites and possibly this often happens since rats with scars usually show infection while those without scars usually do not show infection. The Japanese workers demonstrated that the bite of an infected rat produced the disease in guinea pigs. Another method may be by ingestion, as eating a dead infected rat. It has been shown experimentally that this is possible. Since the spirochetes are quite abundant in the urine of infected rats, it is not improbable that contamination of food stuffs may transmit the organism to other rats. Possibly there are other obscure ways. Some investigators believe that rats are infected by dirt and ditch water in a way similar to human infections, directly through the skin possibly, and are therefore only innocent and harmless carriers of the spirochetes rather than the reservoir of infection.

Rabbits and young dogs are susceptible to infection, but guinea pigs give the best results as experimental animals. The intraperitoneal injection of liver or kidney emulsions from rats produces death in guinea pigs in eight or ten days, while the injection of a liver emulsion from such a guinea pig into another results in death in six or seven days. The animals become markedly jaundiced. Hemorrhages in the large and small intestines appear as pin points, while they are numerous in the lungs. The lymph nodes are usually large and hemorrhagic. The liver shows areas of focal necrosis while the kidneys are greatly engorged with blood.

The Disease in Man

Spirochetal jaundice in man is an acute infection marked by great prostration, headache, vomiting and chills. After an incubation period of about a week, symptoms appear rather suddenly. Towler and Walker (20) divide the disease into three stages. The febrile stage lasts from the first to the fifth day, during which time the spirochetes are present in the blood stream. The icteric stage extends from the sixth to the thirteenth day when the fever decreases but hemorrhages may appear. This is the fatal period. The convalescent period begins at the fifteenth day and lasts to recovery. Jaundice does not appear in all cases. Stokes and his co-workers in Flanders did not find it in more than 60 per cent of cases.

The mortality in Japan was from 11 to 48 per cent of cases. In Europe it varied from 5 to 18 per cent. In America 2 of the 10 known cases died.

The organism *Leptospira icterohemorrhagiae* was found in the organs of persons dying with infectious jaundice by Inada and his associates in Japan, by Hübener and Reiter during the war among German soldiers, by Stokes and Ryle among British soldiers in

Belgium, by Martin and Petit (21) and others among French soldiers in France, and by Monti among Italian soldiers in Italy. In North America among thousands of cases of infectious jaundice studied, the spirochete has been demonstrated only ten times. Efforts of Hiscock and Rogers (23) in the New Haven outbreak, of Symmers (24) in the New York City studies and of Wadsworth and his co-workers in New York State (with one exception), as well as many others, have resulted in failure. Our own efforts in Illinois have been equally unsuccessful.

CASES OF SPIROCHETAL JAUNDICE IN NORTH AMERICA CONFIRMED BY LABORATORY EXAMINATION

Case 1. 1922. Laboratory worker, Albany, New York.
Pricked finger with needle of syringe containing *Leptospira icterohemorrhagiae,* confirmed by guinea pig inoculation of blood culture (14).

Case 2. 1924. Man, age 43, New York State.
Slept in kitchen infested by rats. Fatal case. Sections of liver and kidney showed *Leptospira icterohemorrhagiae* (25).

Case 3. 1925. Man, age 51, Camden, New Jersey.
Diagnosis confirmed by dark field examination of blood (26).

Case 4. 1925. Man, age 44, Philadelphia.
Lived in cheap lodging houses. Diagnosis confirmed by guinea pig inoculation of urine (26).

Case 5. 1926. Man, age 42. Ditch digger, Philadelphia.
Fatal case. Diagnosis confirmed by dark field examination of blood on sixth day and guinea pig inoculation on seventh day (27).

Case 6. 1926. Man, age 48, Toronto, Canada.
Worked in slaughter house. Fatal case. *Leptospira icterohemorrhagiae* found in section of kidney (28).

Case 7. 1926. Man, age 31, Washington, D.C.
Salesman in meat and vegetable section of a market. Diagnosis confirmed by guinea pig inoculation of blood culture (20).

Case 8. Man, age 35, New York City.
Sewer cleaner. Spirochetes demonstrated in urine by animal inoculation and by culture (31).

Case 9. 1926. Man, age 37, New York City.
Laborer. Spirochetes demonstrated in urine and in spinal fluid by animal inoculation and by culture (31).

Case 10. 1926. Man, age 31, Virginia.
Spirochete demonstrated by guinea pig inoculation (22).

Laboratory workers have been infected on several occasions. Besides the one which Wadsworth reported in New York State in 1922, five instances have been reported by Zeulzer in France and Germany and two by Adamski in Warsaw (20).

The manner in which man becomes infected is not altogether clear. It is unlikely that the infection is passed from man to man. On the other hand the contact between rat and man is not necessarily very close. The spirochete when excreted in the urine of the rat may contaminate food, water and soil. Ingestion of the organism is one possibility; another is the passage through the unbroken skin from contaminated surroundings. This explains the infection of ditch diggers and sewer workers, as well as the soldiers in the trenches during the World War.

In England recently an outbreak of 18 cases among coal miners was reported by Gulland and Buchanan (10). One out of every three rats examined showed infection. Further study by Buchanan (29) however revealed that the habitat of the organism was in the roof slime of the coal mine. The slime produced by the glutinous envelope of the organisms formed a jelly-like or zoogleal mass. In this the spirochetes multiplied in great numbers symbiotically with other organisms. When injected into guinea pigs they produced typical symptoms of the disease.

Prevention

The prevention of spirochetal jaundice is a sanitary measure. Those persons who must work in ditches and sewers should protect themselves by boots or shoes. The Japanese workers empha-

Table 37

PER CENT OF RATS INFECTED IN DIFFERENT LOCALITIES

Year	Investigator	Place	Rats found infected
			per cent
1917	Ido (6)	Japan (coal mines)	40
1917	Stokes, Ryle and Tytler (7)	Belgium (war trenches)	40
1919	Foulerton (9)	London	4
1917	Courmont and Durand (33)	France	8
1918	Coles (8)	England	9
1917	Noguchi (4)	New York	7
1917	Jobling and Eggstein (13)	Nashville	10
1922	Wadsworth (14)	Albany	17
1924	Robinson (15)	Baltimore	7
	Neil (16)	Washington	10
1919	Otteraaen (17)	Chicago	6

sized the extermination of rats and field mice as an important prophylactic measure.

A vaccine has been prepared by Noguchi (28), consisting of an emulsion of killed cultures, three injections of which gives complete immunity in guinea pigs. There is no record of its use in man, however. An immune serum has been prepared by the Japanese workers which has been employed for treatment with favorable indications. Kaneko and Okudo report that it possesses a spirochetocidal and spirochetolytic action, destroying the organisms in the tissues, with the possible exception of the kidney.

Items of Note

1. Spirochetal jaundice is caused by *Leptospira icterohemorrhagiae* which is capable of passing through the uninjured skin.
2. The natural habitat of the spirochete is in the rat.
3. Rats in many parts of the world have been found affected.
4. The spirochete seems to inconvenience the rat not at all.
5. Spirochetal jaundice is a new disease, dating from 1916.
6. Spirochetal jaundice must be differentiated from other forms of infectious jaundice which are more common but caused by some other agent.
7. In the United States only nine cases of authentic spirochetal jaundice have been reported in the literature.

BIBLIOGRAPHY

1. Blumer, George
Jour. Am. Med. Asso., 1923, *81*, 353-358.
2. Inada, R., Ido, Y., Hoki, R., Keneko, R., and Ito, H.
Jour. Exp. Med., 1916, *23*, 377-401.
3. Hübener, H., and Reiter, H.
Deutsch. med. Wochenschr., 1916, *42*, 1.
4. Noguchi, H.
Jour. Exp. Med., 1917, *25*, 755-763.
6. Ido, Y., Hoki, R., Ito, H., and Wani, H.
Jour. Exp. Med., 1917, *26*, 341-353.
7. Stokes, A., Ryle, J. A., and Tytler, W. H.
Lancet, 1917, *1*, 142.
8. Coles, A. C.
Lancet, 1918, *1*, 468.
9. Foulerton, A. G. R.
Jour. Path. and Bact., 1919-20, *23*, 78.

10. Gulland, G. L., and Buchanan, Geo.
Brit. Med. Jour., 1924, *1,* 313-314.
11. Noguchi, H.
Jour. Exp. Med., 1919, *30,* 95.
12. Noguchi, H.
Jour. Exp. Med., 1918, *27,* 609-625.
13. Jobling, J. W., and Eggstein, A. A.
Jour. Am. Med. Asso., 1917, *69,* 1797.
14. Wadsworth, A. B., Langworthy, H. V., Stewart, F. C., and Moore, A. C.
Jour. Am. Med. Asso., 1922, *78,* 1120-1121.
15. Robinson, G. H.
Am. Jour. Hyg., 1927, *4,* 327-329.
16. Neill, M. H.
Public Health Reports, U. S. Public Health Service, 1918, *33,* 717.
17. Otteraaen, A.
Jour. Inf. Dis., 1919, *24,* 485.
18. Noguchi, H.
Jour. Exp. Med., 1919, *29,* 547.
19. Theiler, M., and Sellards, A. W.
Am. Jour. Trop. Med., 1926, *6,* 383.
20. Towler, Harry H., and Walker, John E.
Jour. Am. Med. Asso., 1927, *89,* 86-89.
21. Martin, A., and Petit, L.
Presse Med., 1916, 569.
22. Mulholland, H. B., and Bray, W. E.
Jour. Am. Med. Asso., 1928, *90,* 1113.
23. Hiscock, Ira V., and Rogers, O. F.
Jour. Am. Med. Asso., 1922, *78,* 488-490.
24. Symmers, Douglas
Jour. Am. Med. Asso., 1920, *74,* 1153.
25. McDowell, E. S.
New York State Jour. Med., 1925, *25,* 19.
26. Sailor, Joseph
Am. Jour. Med. Sci., 1925, *170,* 332.
27. Hayman, J. M., and Lynch, F. B.
Am. Jour. Med. Sci., 1927, *173,* 80-89.
28. Bates, J. E.
Jour. Canadian Med. Asso., 1926, *16,* 1421.
29. Buchanan, Geo.
Brit. Med. Jour., 1924, *2,* 990-993.
30. Noguchi, H.
Jour. Exp. Med., 1918, *28,* 561.
31. Cushing, E. H.
Jour. Am. Med. Asso., 1927, *89,* 1041.

32. Pagniez, P., Cayrel, A., Lévy, P. P., and de Léobardy, J.
Bull. de la Soc. Med. des Hop., 1918, *42,* 31-36.

33. Courmont, J., and Durand, P.
Bull. et. mem. Soc. méd. hop., Paris, 1917, *33,* 115, 3rd. Ser.

34. Walch, E. W., and Walch-Sorgdrager, G. B.
Am. Jour. Hyg., 1927, *7,* 393-406.

Chapter XVII

RAT-BITE FEVER

Rat-bite fever is primarily an affection of rats; secondarily man is infected following the bite of a rat or sometimes of a weasel, ferret, squirrel or cat.

The term "sodoku" is common for the disease in Japan and is found in English and American literature.

Sodoku has been known in Japan for many years, being familiar both to physicians and laity. In 1900 Miyake (1) brought the disease to the attention of European investigators, who up to this time had quite ignored it, there being references to only two cases in European literature.

In England the first cases were reported by Horder in 1909, since which time the disease appears to have been quite prevalent. In France, Spain, Italy, Germany and other continental European countries a few reports have been received from time to time.

In America rat-bite fever has been known for nearly a century. Wilcox (2) in 1839 reported the first case in a man forty years old. The next year Watson (3) gave an account of a seaman, bitten by a rat over the eyebrow, who developed the disease. In 1869 there was a mother and son reported by Gilliam (4) who developed the disease within two days of each other. Three reports appeared in 1872 by Packard (5) in Philadelphia and Earle (6) and Reece (7) in Chicago. Cook (8) in 1885 had one case and Banker (9) in 1886 four cases. In 1901 Evans (10) published an account of three cases. Two girls were bitten by the same rat at the same time, both of whom took sick on the same day three weeks later. Proescher (11) in 1912 added another.

In 1915 Crohn (12) collected histories on 24 Japanese cases, 16 American, 8 English, 2 French, 1 Italian and 1 Indian. The next year Blake (13) added 28 more histories making 81 in all. Since that time the disease has been reported with increasing frequency from many parts of the world.

During the ten years from 1918 to 1927 there were reported in the scientific literature 90 cases as follows:

England	12	Italian Somoliland	1
France	8	New Guinea	1
United States	8	Austria	2
Australia	2	Switzerland	1
India	4	China	3
Italy	15	Mexico	1
Spain	1	Hawaii	1
Russia	2	Brazil	6
Holland	2	British Guinea	1
Germany	3		

The disease seems to be uninfluenced by climatic or regional conditions.

THE ETIOLOGICAL AGENT

Several organisms have been reported as causing rat-bite fever, *Sporozoa muris* by Ogata, a diplococcus by Middleton, a poorly staining bacillus by Proescher, *Micrococcus tetragenous* by Ofenheim, a streptococcus by Douglas and *Streptothrix muris ratti* by Schottmüller. In 1916 Futaki and his Japanese associates described a spirochete which they named *Spirocheta morsus muris.* The organism has been found in the wound at the site of the bite, in the lymph and salivary glands and in the circulating blood. While further confirmation is necessary, it has been generally accepted tentatively that this organism is the cause of rat-bite fever, at least as it exists in Japan. The other organisms described may be considered possibly in the light of secondary invaders.

THE DISEASE IN ANIMALS

Infection of rats with *Spirocheta morsus muris* does not seem to inconvenience them. The Japanese workers (14) did not find the organism present in the blood of all rats and there was no relation between the kind of rat and the ratio of infection. They found the organism only in the blood stream and not in the saliva and it was suggested that possibly transmission of the spirochete from rat to man occurs from infection of the wound through bleeding gums. Later investigation, however, showed the spirochetes present in the saliva and it is undoubted that the spirochete is conveyed in this way. About 3 per cent of brown rats have been found infected. Leadingham (15) found 73 rats in Atlanta, Georgia, to be free of infection.

Rat-bite fever following the bite of a cat has been reported in several instances (16) while Nixon (17) describes the same condition following the bite of a ferret and Schottmüller (18) the bite of a squirrel. The weasel has likewise been implicated by Dick and Tunnicliff (19) as well as the dog. Such animals must be considered probably as mechanical carriers.

The Disease in Man

Rat-bite fever in man is characterized by a local inflammatory condition about the site of the bite-wound with enlargement of the contiguous lymphatic glands, and accompanied by paroxysms of fever followed by periods of remission.

Crohn (12) studied a large number of cases, finding the incubation period to average twelve days. The more severe the bite, however, the shorter the incubation period and the more severe the illness. Cases in which the incubation period lasted from 1 to 5 days, the illness endured 111 days; incubation period 6 to 15 days, illness 50 days; incubation period 16 to 35 days, illness 54 days.

Occupation, age and sex play no part in susceptibility to infection but are of some significance in the possibility of exposure. Crohn reported 31 males to only 20 females. Adults were more often infected, especially men and grown boys in such occupations as seamen and farmers.

The location of the wound in 52 cases was on the hand and arm 36 times, face and head 11, leg and foot 3 and not noted 2. As in rabies, a bite on an exposed surface was more apt to result in infection than on a protected surface where the clothing might wipe off the organism from the teeth. The bite of an infected animal does not invariably result in infection, since there are several instances of two persons being bitten by the same rat and only one contracting the disease.

The cure of syphilis by rat-bite fever has been accomplished experimentally in a number of institutions. The patient is infected by injection of a small amount of blood from a white rat which is harboring the spirochetes. When the syphilis is cured, the rat-bite fever is cured by an injection of arsphenamine. The severe reactions from the rat-bite fever have tended to limit the use of the method however.

Prevention and Control

The prevention of rat-bite fever is accomplished by the various methods which have been devised for the suppression and destruction of rats. Prophylactic treatment of persons who have been bitten consists in prompt cautery of the wound with antiseptic dressings. The cure of the disease has been rendered more certain by the use of arsphenamine, one injection of which is usually sufficient.

ITEMS OF NOTE

1. Rat-bite fever is probably caused by *Spirocheta morsus muris.*
2. The spirochete causes no inconvenience in rats.
3. Human cases have been reported in America for nearly a century.
4. The disease in man is contracted by the bite of an infected rat, rarely by other animals.
5. The prevention of the disease consists in the elimination of rats.

BIBLIOGRAPHY

1. MIYAKE, H.
   Mitt. a.d. Grenzgeb. d. Med. u. Chir., 1900, *5,* 231.
2. WILCOX, W.
   Am. Jour. Med. Sci., 1840, *26,* 245.
3. WATSON, J.
   New York Jour. Med. and Surg., 1840, *3,* 363.
4. GILLIAM, D. I.
   Cincinnati Lancet and Observer, 1869, *12,* 16.
5. PACKARD, J.
   Philadelphia Med. Times, 1872, *2,* 408.
6. EARLE, C. W.
   Chicago Med. Examiner, 1872, *13,* 3.
7. REECE, M.
   Chicago Med. Examiner, 1872, *13,* 51.
8. COOK
   Indiana Med. Jour., 1912, *2,* 77.
9. BANKER, A. J.
   Am. Pract. and News, 1886, N.S. *11,* 70.
10. EVANS, W. A.
    Tr. Chi. Path. Soc., 1901-1903, *5,* 298.
11. PROESCHER, F.
    Intern. Clinics, 1912, *4,* Series 21, 77.
12. CROHN, B. B.
    Arch. Int. Med., 1915, *15,* 1014-1039.
13. BLAKE, F. G.
    Jour. Exp. Med., 1916, *23,* 39-60.
14. FUTAKI, K., TAKAKI, F., TANIGUCHI, T., and OSUMI, S.
    Jour. Exp. Med., 1917, *25,* 33-34.

15. Leadingham, R. S.
Jour. Am. Med. Asso., 1927, *89,* 239.

16. Nejrotti, G. M.
Abstract Jour. Am. Med. Asso., 1926, *87,* 825.
Giglioli, G. O. M.
Trans. Roy. Soc. Trop. Med. and Hyg., 1927, *20,* 504-505.

17. Nixon, J. H.
Brit. Med. Jour., 1914, *2,* 629.

18. Schottmüller, H.
Dermat. Wchschr., 1914, *58,* Suppl. 77.

19. Dick, G. F., and Tunnicliff, Ruth
Jour. Inf. Dis., 1918, *23,* 183.

CHAPTER XVIII

ROCKY MOUNTAIN SPOTTED FEVER

Rocky Mountain spotted fever is regarded as primarily an affection of small rodents; it is transmitted to man by means of infected ticks.

The disease has been known by numerous other designations such as mountain fever, bull fever, spotted fever, black fever, blue disease and typho-malaria fever of the Rocky Mountains.

HISTORY

Rocky Mountain spotted fever has existed in the northwestern United States since that section was first settled by the white man and there is no reason to suppose that the Indians did not have the affection before that time, though there are no authentic records. Fricks states however that the early settlers in the Rocky Mountains reported a tradition which existed among the Indians of the region that some sections of the mountains could not be visited in certain seasons of the year without engendering the wrath of the Great Spirit.

Beginning with the immigration of the Mormons in 1847 and continuing during the period of the "gold rush" to California, vague descriptions are given of troubles that may well have been Rocky Mountain spotted fever, but at that time and in the years that followed it was confused with other better known diseases.

Wilson and Chowning (2), making an investigation in 1902 for the Montana State Board of Health, stated that spotted fever had been known for twenty years in Idaho, while the first case in the Bitter Root Valley occurred in 1873. There were very few white men in the Bitter Root Valley at that time. The Indians inhabited the valley until 1890 but there was no record of the disease among them. They further state that "the disease has been recognized as a clinical entity for fifteen or twenty years" but the only previously published article was that of Maxey (1) in 1899. In 1896 Major M. W. Wood (4) made an unpublished report of the disease to the Surgeon General of the United States Army. To Maxey however goes the credit of

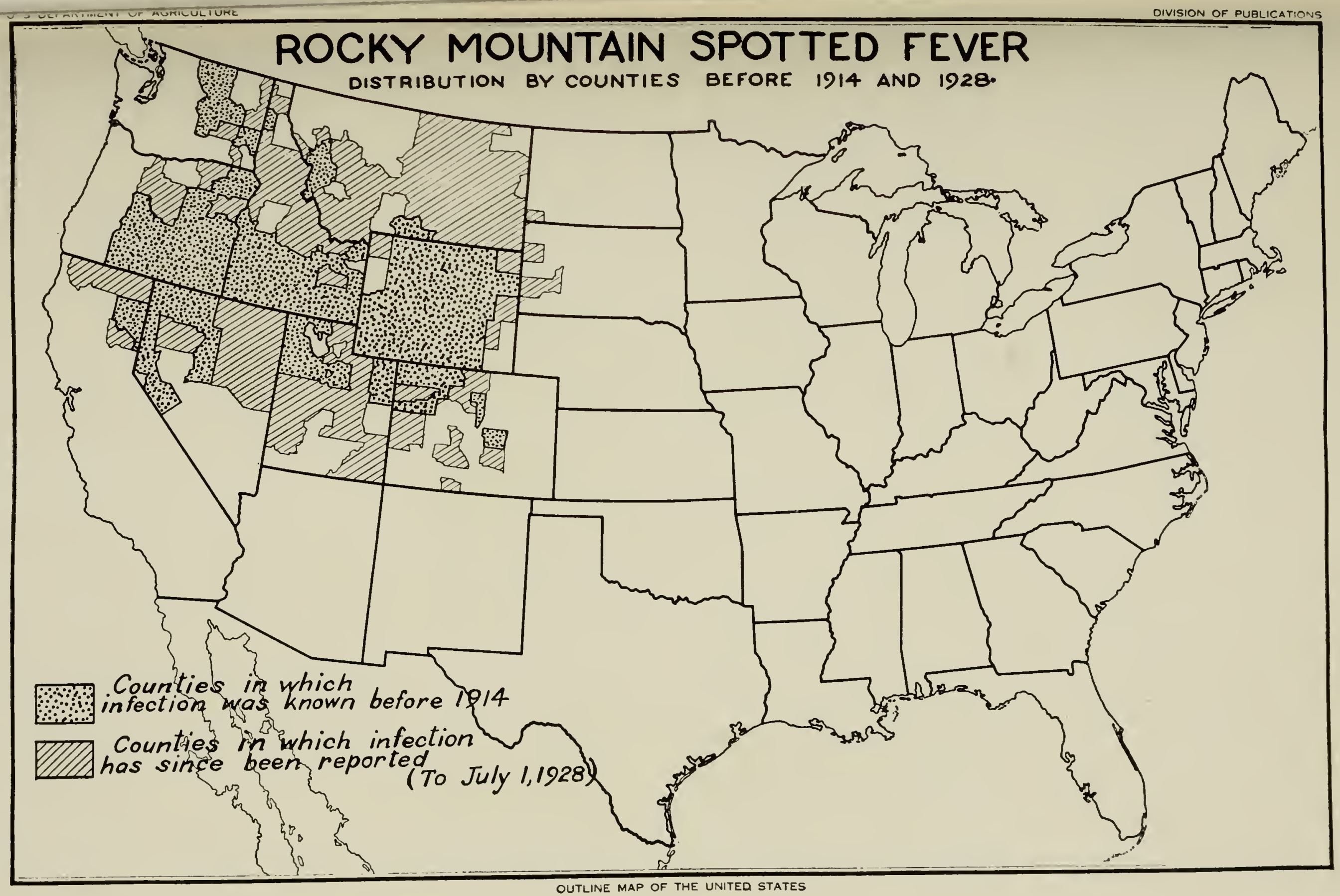

U S DEPARTMENT OF AGRICULTURE
DIVISION OF PUBLICATIONS
ROCKY MOUNTAIN SPOTTED FEVER
DISTRIBUTION BY COUNTIES BEFORE 1914 AND 1928.
Counties in which infection was known before 1914
Counties in which infection has since been reported (To July 1, 1928)
OUTLINE MAP OF THE UNITED STATES

Fig. 29.

bringing Rocky Mountain Spotted Fever to the attention of the medical world as a new and distinct disease, differentiated from other infections.

The United States Public Health Service began investigations in 1904 and have continued them since that time at the Hygienic Laboratory in Washington and since 1922 at the branch laboratory at Hamilton, Montana.

GEOGRAPHICAL DISTRIBUTION

Rocky Mountain spotted fever is known to exist only in the United States. It is practically confined to nine of the northwestern mountain states as follows: Washington, Oregon, California, Idaho, Nevada, Utah, Montana, Wyoming, and Colorado. A few cases have also been reported from the extreme western portions of North and South Dakota. There are certain endemic areas in each of these states where the disease recurs annually, while other large areas surrounding them seem to remain free. Ordinarily as land is cleared and taken up for farming purposes, the disease disappears from the endemic foci.

SEASONAL PREVALENCE

The spring and early summer months are the periods when most of the cases of Rocky Mountain spotted fever occur, corresponding to the active feeding period of the wood tick. In the colder climate of the high mountains the tick may feed late into the summer thus causing some cases as late as August.

Wilson and Chowning divided 126 cases studied by them as follows:

March	6 cases
April	24 cases
May	46 cases
June	35 cases
July	5 cases
Spring (exact month not known)	10 cases
	126 cases

THE CAUSATIVE AGENT

The virus of Rocky Mountain spotted fever is present in the blood stream of infected individuals and animals and can be transferred by injections from animal to animal indefinitely (Ricketts). In fact, this is the usual method of propagating the virus under laboratory conditions, for it can be cultivated artificially, only with great difficulty.

Structures in infected blood were discovered by Ricketts, who believed them to be the causative agent of the disease. Wolbach (5) has made a study of these bodies, designating them *Dermacentroxenus rickettsi.* They occur in infected ticks in different forms as (a) an intracellular bacillus-like form, without chromatid granules, present only in the cytoplasm of the alimentary tract cells; (b) a relatively small rod-shaped form with chromatid granules, seen within the cells of many tissues, and within the nuclei of the cells of many tissues; and (c) a relatively large, lanceolate paired form, which persists in the tissues of ticks and notably in the salivary glands. This latter form is easily demonstrated in human and animal tissues and in tick eggs by the usual staining methods.

Wolbach and Schlesinger (6) cultivated the organisms artificially in virulent form in tissue plasma cultures, keeping them alive for four weeks by transferring the tissue to fresh plasma.

Wolbach believes that *Dermacentroxenus rickettsi* is the cause of Rocky Mountain spotted fever because:

1. It is constantly present in lesions characteristic of the disease in man, monkey, rabbit and guinea pig.
2. Its constant development and exhibition of definite morphological sequence in ticks fed on infected animals and the absence of similar micro-organisms in ticks proved to be non-ineffective.
3. The successful cultivation of the micro-organism in virulent form in tissue plasma cultures.

These observations have not been entirely in accord with those of Parker and Spencer (7), who found a few apparently normal ticks harbouring rickettsia bodies indistinguishable from those causing the disease, while a few infected ticks failed to show such bodies. In a later work these authors (8) make the suggestion that "the virus of Rocky Mountain spotted fever may assume a form incapable of demonstration by known methods." They found that the virus will not pass through Berkefeld "N" and "V" filters, coarse enough to allow the passage of staphylococci. The failure to pass the filter was not due to a chemical affinity for or adsorptive property of the material of which the candles were made.

TICKS

The transmission of Rocky Mountain spotted fever from animal to animal or from animal to man may take place only by means of the tick. The following ticks have been found naturally infected in nature:

Dermacentor andersoni
Dermacentor modestus
Dermacentor occidentalis
Haemaphysalis leporis-palustris

In addition, the eastern ticks *Dermacentor variabilis* and the closely related *Amblyomma americanum* have been experimentally infected as well as *Dermacentor albipictus.* No other biting arachnid and no biting insect has been found capable of transmitting the virus. The bed bug, the stable fly, the fowl tick, the monkey louse and the guinea pig louse all have given negative results. The virus will live longer in the body of the bed bug than it will live at room temperature, however.

The rôle of the tick in transmitting the disease was first advanced by Wilson and Chowning (2) in 1902 and was fully confirmed by Ricketts (3) in 1906. Once the tick becomes infected it remains so for life and is capable of passing the infection on through the egg.

The number of infected ticks in nature, based upon the frequency of human infections following tick bites, has been estimated at ten per cent. The percentage is much lower than this, however, in experiments where ticks have been examined. Among 2,265 ticks allowed to feed on guinea pigs only 8 were found capable of infecting the animals, 0.28 per cent. Ricketts found one in 296, Maver 2 in 402 and McClintic 6 in 1,037.

THE DISEASE IN ANIMALS

The following rodents have been found susceptible to the virus of Rocky Mountain spotted fever.

Columbian ground squirrel—*Citellus columbianus.*

Side-stripped ground squirrel—*Callospermophilus lateralis cinerascens.*

Pine squirrel—*Sciurus hudsonicus.*

Yellow bellied chipmunk—*Eutamias luteiventris.*

White bellied chipmunk—*Eutamias quadrivittatus umbrinus.*

Wood-chuck—*Marmota flaviventor.*

Badger—*Taxidea taxus.*
White tailed jack-rabbit—*Lepus campestris.*
Cottontail rabbit—*Sylvilagus nuttalli.*

They are not seriously inconvenienced by the affection, but it is assumed act as passive carriers. Their relative importance depends upon their abundance in different localities and their selections as hosts for ticks to feed upon.

Cattle, sheep, goats and old horses are immune, as well as the wild large animals of the endemic centers, so far as tested. Ricketts succeeded in infecting young horses by experimental inoculation. White mice cannot be infected.

Man, rhesus monkeys, guinea pigs and white rats are highly susceptible. Domestic rabbits are somewhat susceptible.

The Disease in Man

Rocky Mountain spotted fever in man is characterized by a petechial eruption beginning first on the extremities and continuing over the body, accompanied by headaches, muscular pains and fever. Man takes no part in the spread of the disease, contracting it by the bite of an infected tick, but transmitting it no further either directly or by way of other ticks.

The number of cases occurring annually is about five hundred, and is decreasing each year due to reclamation of the land for farming purposes with consequent reduction of ticks. The greatest number of cases reported in any one year has never amounted to a thousand.

The mortality on the west side of the Bitter Root Valley is about 90 per cent, while in Idaho it does not reach 5 per cent. Outside the Bitter Root Valley and one or two small areas in Wyoming, the mortality is generally fairly low. Several investigators have laid down their lives in the study of the disease, among them being four from the United States Public Health Service—Passed Assistant Surgeon T. B. McClintic and Laboratory Assistants William Gettinger, G. W. Cowan and A. Leroy Kerlee.

The disease is rural in character rather than urban. In almost every case the patient gives a history of having visited a tick-infested region within the last two weeks or less. Because of the habits of the tick, which is necessary for the infection of man, it would be impossible for the disease to exist to any extent either in cities or well-farmed rural areas.

PREVENTION

Several methods of prevention of Rocky Mountain spotted fever have been emphasized by different workers—tick eradication, avoiding tick bites and immunization.

The United States Public Health Service, together with the United States Department of Agriculture, have studied methods of tick eradication. Fricks recommends the destruction of small, wild animals upon which the immature ticks feed and the prohibition of horses and cattle upon which the adult ticks feed from tick-infested regions during the tick feeding season. The clearing, fencing and cultivating of arable lands will cause ticks to disappear in a few years. Fricks believes that sheep pick up more ticks than do any other domestic animal and that by feeding them away from the settlements into the mountains each year they mechanically sweep the ticks into the remote places of the mountains. Sheep graze close thus reducing tick breeding by destroying shelter for ticks as well as for the small animals upon which the ticks feed.

The surest way to prevent human infection is to avoid infested regions during the tick feeding season. If it is necessary to go into those regions, clothing fitting properly about the legs, arms and neck will prevent, to a certain extent the entrance of ticks. It is necessary however to strip off the clothing every few hours and make a careful inspection.

Vaccination against Rocky Mountain spotted fever has been advocated by Spencer and Parker (9). Ticks are allowed to feed upon infected guinea pigs until well infected. They are again fed after an interval of 20 days or more and then eviscerated and ground up and an emulsion prepared containing 0.5 per cent phenol. This is the only vaccine made from an insect host for human use. In 1924, after thorough animal experimentation, Dr. R. R. Spencer took the first human dose without untoward results. In 1926 among 300 sheepherders in the Shoshone District of Idaho, 140 were vaccinated. None developed the disease while among the 160 unvaccinated there were 8 cases and among other unvaccinated persons in the same district 25 cases. In the Bitter Root Valley 600 persons were vaccinated without contracting the disease.

Among laboratory workers there were five virulent and fatal infections before the vaccine was discovered. Since that time there have been four very mild cases among vaccinated workers.

One of these latter was a man 62 years old infected with the fatal Bitter Root strain—the only case on record where a person of this age recovered from this strain of virus.

ITEMS OF NOTE

1. Rocky Mountain spotted fever is a disease confined to the northwestern portion of the United States.
2. The nature of the causative agent has not been definitely determined, although several workers have sacrificed their lives in the attempt.
3. Small animals are necessary for the immature ticks to feed upon, while large animals or man are victims of the adult ticks.
4. The wood tick *Dermacentor* is the agent by which the disease is spread from animal to animal and from animal to man.
5. Ticks once infected remain so for life and pass the virus to the succeeding generation.
6. The disease is not transmitted from person to person, either directly or by other means.
7. The disease is rural in nature and cannot exist to any extent either in cities or well-developed farming communities.
8. A vaccine which has given encouraging results has been prepared for the protection of exposed individuals.

BIBLIOGRAPHY

1. Maxey, E. E.
Med. Sentinel, Portland, Ore., 1899, *7,* 433-438.
2. Wilson, L. B., and Chowning, W. M.
Jour. Am. Med. Asso., 1902, *39,* 131-136.
3. Ricketts, H. T.
Jour. Am. Med. Asso., 1906, *47,* 358.
4. Wood, M. W.
Rep. of Surg. Gen. U. S. Army, 1896, 60-65.
5. Wolbach, S. B.
Jour. Med. Res., 1919, *36,* 1-197.
6. Wolbach, S. B., and Schlesinger, M. J.
Jour. Med. Res., 1923, *39,* 231-256.
7. Spencer, R. R., and Parker, R. R.
Public Health Reports, 1924, *39,* 3027-3040.
8. Spencer, R. R., and Parker, R. R.
Public Health Reports, 1924, *39,* 3251-3255.
9. Spencer, R. R., and Parker, R. R.
Public Health Reports, 1925, *40,* 2159-2167.

SECTION THREE

HUMAN DISEASES SPREAD BY ANIMALS

SECTION THREE

HUMAN DISEASES SPREAD BY ANIMALS

CHAPTER XIX

THE RELATION OF HUMAN INFECTIONS TO ANIMALS

The Committee on the Control of Communicable Diseases of the American Public Health Association has published a list of forty-two communicable diseases to which man is subject. Of this number there are twenty-six to which animals are quite refractory and have no part in the dissemination of the organisms causing the disease.

Acute infectious conjunctivitis
Ancylostomiosis (hookworm)
Chicken pox
Cholera
Dengue
Dysentery (amoebic)
Dysentery (bacillary)
Epidemic (lethargic) encephalitis
Favus
German measles
Gonorrhea
Influenza
Leprosy
Malaria
Measles
Meningococcus meningitis
Mumps
Paratyphoid fever
Pneumonia (acute lobar)
Poliomyelitis
Syphilis
Trachoma
Typhoid fever
Typhus fever
Whooping cough
Yellow fever

Epidemiological evidence from time to time seemed to implicate animals in the spread of some of these infections. Poliomyelitis has been associated with dogs, cats, chickens, and colts in various reports. I investigated an epidemic of paralysis among hogs on a farm where two children were sick with poliomyelitis, but histological examination of the spinal cord of the hogs revealed no evidence to indicate that the affection was poliomyelitis. Two colts on another farm where poliomyelitis existed in the family were kept under observation for several weeks, but the so-called paralysis apparently was due to a nutritional deficiency. Frost (2) investigated the connection of paralyzed dogs, chickens, and rabbits to the epidemics of poliomyelitis in Iowa in 1910 and in Cincinnati, Ohio, in 1911, finding no evidence to support the contention that such animals were suffering from the disease or acting as carriers.

Poliomyelitis has been transmitted through cow's milk, but there is no reason to believe the cow had any connection with the contamination of the milk.

Typhoid fever transmitted by the hen through the egg has been suggested as a possibility. It is known that poultry will readily partake of human feces if given the opportunity and this happens on occasions too numerous, around poorly protected privies and out-houses. Mitchell and Bloomer (1), however, found hens entirely refractory to the typhoid bacillus both by injection and ingestion, nor was a "carrier" state set up. There is no evidence to believe that the hen is capable of transmitting typhoid through the egg.

Typhoid fever is often transmitted through cow's milk, but the cow is not responsible, even though she partakes of badly infected drinking water. Contamination of the milk in every instance arises directly from a human source.

There are ten diseases reported by the Committee on the Control of Communicable Diseases which are primarily animal in origin. Man is subject to infection usually directly from an animal and rarely passes the infection on to another person. Isolated instances can be enumerated where some of the diseases have been passed from person to person. In the pneumonic form of plague such direct contact is the usual form of dissemination. Tuberculosis other than pulmonary includes the bovine and avian types, but may also include the human type.

Actinomycosis
Anthrax
Glanders
Undulant fever
Plague
Rabies

Rocky Mountain spotted fever
Trichinosis
Tuberculosis (other than pulmonary)
Tularemia

The above diseases are discussed in separate chapters. In addition to that list there are several diseases of animal origin of minor epidemiological importance to man which are also discussed.

Foot-and-mouth disease
Milk sickness
Psittacosis
Infections with animal parasites (other than trichina)
Spirochetal jaundice
Rat-bite fever
Food infections (Salmonella group)

Tetanus is an infection which cannot be charged directly to animals except as they act as passive carriers of the organisms, disseminating them almost universally. Man becomes infected, as well as do animals, through contamination of a wound with dirt-bearing tetanus spores. Botulism and gas gangrene, not mentioned in the committee report, are in the same class as tetanus.

There are five diseases reported by the committee which are primarily human in origin but to which certain animals are susceptible. They are of importance because infected animals may pass them back again to man. They are as follows:

Diphtheria
Septic sore throat
Scarlet fever
Smallpox
Tuberculosis (human type)

BIBLIOGRAPHY

1. Mitchell, O. W. H., and Bloomer, G. T.
Jour. Med. Research, 1914, *31*, 247.
2. Frost, W. H.
U. S. Public Health Service, Hyg. Lab. Bull. 90, 1914.

CHAPTER XX

SEPTIC SORE THROAT

Septic sore throat in man is caused by *Streptococcus epidemicus.* The organism is primarily a human pathogen, but it may set up a focus of infection in the udder of the cow, from whence it is transmitted back to man by way of the milk supply.

HISTORY

Septic sore throat due to infected milk has been recognized in Great Britain for many years. In 1875 there occurred at South Kensington, England (1), several cases of sore throat and scarlet fever among 20 persons who had used cream coming from a district where there had been 119 cases of sore throat. It is impossible to determine from the evidence presented whether this was a scarlet fever outbreak or a mixed infection of scarlet fever and septic sore throat.

The first authentic record of an epidemic of the tonsilitis or quinsy type is that at Aberdeen (2). Among 110 families supplied by a single dairy, 90 families were affected, with 300 cases. During the following years similar epidemics occurred with considerable regularity, the milk supply being incriminated by circumstantial evidence only.

The first outbreak of septic sore throat recognized in the United States occurred in Boston, Massachusetts, in May, 1911. Winslow (3) made a careful epidemiological study of the 1,400 cases involved, finding that 70 per cent were supplied with milk from one dairy, the cases exactly coinciding with the two main delivery routes of that dairy. The symptoms all pointed to a streptococcus as the infecting agent but there was little definite information available on his point. In the laboratory of the Boston Board of Health as well as other laboratories no constant organism was found. Theobald Smith, however, isolated streptococci from four cases at autopsy.

The relationship of streptococci to udder infections of cows was suspected as early as 1884 by Nocard and Mollerau (4). In 1889 Bang produced inflammation of the udder of a cow by injecting into the milk duct a streptococcus isolated from a case of mastitis in another cow.

The classification of the streptococci was confused, however, and it was not until 1915 that Davis distinguished *Streptococcus epidemicus* from the ordinary bovine streptococci.

Streptococcus Epidemicus

Bergey (25) lists 25 members of the genus *Streptococcus,* 15 of which are parasitic. Only two are of interest in this study—*Streptococcus pyogenes,* a hemolytic organism found in acute pathological conditions of man, and *Streptococcus mastiditis,* the cause of infectious mastitis of cows. *Streptococcus epidemicus* is considered identical with *Streptococcus pyogenes* by Bergey, and is not listed as a seperate species.

Different investigators in times past have applied various names to the streptococci found by them in cases of bovine mastitis, such as *Streptococcus mastitis,* streptococcus of bovine mastitis, *Streptococcus agalactiae contagiosae, Streptococcus pyogenes bovis,* etc. Probably nearly all these organisms were of the same type but could not be properly classified due to the confused methods for differentiating and classifying them.

It was formerly supposed that streptococci causing bovine mastitis were pathogenic for man and routine tests were made in many laboratories for the presence of such organisms. The finding of streptococci of any nature was sufficient evidence to condemn the milk.

Smith and Brown (16) in 1915 separated the hemolytic streptococci found in milk into the alpha type which produced a greenish discoloration with a small amount of hemolysis, and the beta type which produced a sharply defined, clear, transparent, colorless zone of hemolysis 2 to 4 mm. in diameter. Streptococci from the Boston, Chicago, and Baltimore epidemics fell into the latter group.

Davis in 1916 showed that hemolytic streptococci of the beta type occurred commonly in both pasteurized and unpasteurized milk. Strains varied among themselves, were more resistant to heat than human strains, and possessed no virulence for rabbits. There was no reason to attach sanitary significance to these organisms. Occasionally hemolytic streptococci were found in milk which differed from the above groups, resembling instead organisms from human sources. To these he applied the name *Streptococcus epidemicus.*

Frost, Gumm, and Thomas (22) studied the milk from about 1,000 different cows on five certified dairies over a period of one and a half years. The 394 cultures of the true beta type encountered fell into six different groups. The bovine organism, *Streptococcus mastitidis,* was most frequently found—186 times; the remaining bovine organisms were present in decreasing numbers in the following order—*Streptococcus infrequens, Streptococcus subacidus,* and *Streptococcus hemolyticus i; Streptococcus asalignus* was described as a new species. *Streptococcus epidemicus Davis* was found in four animals.

The following method for the detection of *Streptococcus epidemicus* in certified milk was described by Bundesen (23) as having been adopted by the Chicago Health Department.

> Hemolytic organisms found in certified milk should be regarded as of no significance unless conforming to the following characteristics:
>
> They should be streptococci of proper morphology, giving colonies of the beta type on blood agar.
>
> Production of hemolysin should be sufficient to produce full hemolysis of rabbits' blood corpuscles within two hours.
>
> They should kill both of two mice injected, acutely within forty-eight hours, and should be recovered from the animals and identified.
>
> Samples containing organisms conforming to these tests may be regarded as under suspicion, but these data are merely suggestive and not proof that the organism is dangerous to human beings. Any action which may be taken must necessarily be determined by practical circumstances. If, however, an absolute safeguard against the possibility of throat outbreaks is desired, pasteurization or a shut-off of the supply until subsequent samples prove negative is the only reliable procedure.

Brown, Frost, and Shaw (24) state that an organism found in milk must fulfill the following conditions before it can be called *Streptococcus epidemicus*—a streptococcus of round or slightly flattened elements, producing hemolyzed zones of the beta type in blood agar, rapidly hemolyzing blood corpuscles in fluid media, markedly pathogenic for mice, fermenting glucose, lactose, saccharose and salacin but not mannite, producing a final hydrogen ion concentration not above P_h 4.8 in glucose broth, failing to hydrolyze sodium hippurate, and probably always encapsulated when observed under suitable conditions, in the last respect differing from most strains of hemolytic streptococci isolated from sporadic human infections.

Table 38

PREVALENCE OF HEMOLYTIC STREPTOCOCCI IN HIGH GRADE MILK

Frost and Bachman (19)

Herd Number	Number of cows	Alpha colonies	Beta colonies	Total hemolytic colonies
1	124	19	7	26
2	97	29	18	47
3	144	16	7	23
4	47	8	8	16
	412	18%	10%	28%

The Affection in Animals

Mammitis of cows may be caused by a variety of organisms which not only interfere seriously with the milk supply, but also act as a menace to the public health. Savage (7) in England investigated 36 cases of mammitis, finding 68 per cent due to streptococci and another 10 per cent probably streptococci; 16 per cent were staphylococci, 3 per cent colon bacilli, and 3 per cent tubercle bacilli. Carpenter (8) studied 150 samples of milk from infected udders of cattle in 76 herds of cattle in New York, Pennsylvania, and Massachusetts, finding the following causative organisms—hemolytic streptococci 118 times (78.6 per cent), beta hemolytic streptococci 10, *Bacillus pyogenes* 6, *Staphylococcus aureus* 6, *Staphylococcus albus* 3, unidentified Gram negative rod 2, colon bacilli 3, mixed alpha hemolytic streptococci and *Staphylococcus aureus* 1, and negative 1. Hardenbergh and Schlotthauer (9) observed the incidence of mastitis over a period of two and a half years in four herds, comprising about 280 cows. Sixty-eight cases were detected and studied, of which 40 were due to streptococci,, 7 to staphylococci, 6 to *Bacillus coli,* and 1 to *Bacillus lactis aerogenes.* Eight infections were mixed and one was unidentified. In 1914 Davis and Capps (10) applied a hemolytic streptococcus directly to an abrasion on the udder of a cow, causing an infection as evidenced by the enormous number of streptococci found in the milk, but no external sign of inflammation, caking, or "gargety" milk. In another experiment streptococci directly from a case of tonsilitis were introduced into the milk duct, causing an infection of several weeks' duration with numerous streptococci in the milk. Their conclusions, that streptococci of human origin may cause mastitis without external evidence of infection, were soon corroborated by other investigators (21). Smith and Brown (16), as well as Krumwiede and Valentine (12), showed that streptococci commonly associated with bovine mastitis could be differentiated from those causing human tonsilitis.

The usual source of infection of the cow is an infected person. Frost and Carr (26) recite their experience with an epidemic arising from a very high grade milk supply. Laboratory examinations revealed enormous numbers of *Streptococcus epidemicus* —36,000,000 per cubic centimeter—in the milk of one animal. A few days later a second cow was found infected and a little later still a third cow. Cultures from the nose and throat of each person in the dairy revealed that two were carrying *Streptococcus epidemicus.* One was a woman in the house who had no part in handling the milk; the other was the hired man, whose chief duties were the care of the cows and the milk. After he left the dairy, there was no more trouble.

One quarter of the udder may be infected with streptococci without the other quarters becoming involved. It is necessary, therefore, to examine milk from each quarter when looking for infection. Cows once infected with *Streptococcus epidemicus* are likely to remain sources of danger over long periods of time and probably never should be returned to the milking line.

Prevalence of Septic Sore Throat

Although epidemics of septic sore throat of milk origin have been recognized in England for half a century and Swithinbank and Newman (11) in 1903 recorded that hardly a year passed without an outbreak, yet it was not until 1911 that the disease was reported in the United States. Winslow's (3) study of the Boston epidemic focused the attention of sanitarians on this cause of infection with the result that numerous reports of similar outbreaks appeared in the next few years. In December, 1911, and January, 1912, an epidemic occurred in Chicago, Illinois, in which Capps and Miller (12) estimated 10,000 cases were involved, traced to a milk supply supposed to have been pasteurized. Mann (13) encountered an outbreak in Concord, New Hampshire, in January and February, 1912, where 426 persons were made sick by the use of milk and cream from one dairy. In February and March of the same year 602 cases with 28 deaths occurred in an epidemic in Baltimore, Maryland, where Frost (14) found 65 per cent of cases used one milk supply. Veterinarians could find no mastitis in the cows of the suspected herd, and it was thought that streptococci were introduced directly by infected persons. In April and May, 1913, North, White, and Avery (15) studied two epidemics involving 669 cases with 14 deaths which occurred simultaneously in different localities but having their source in the same dairy. Two cows were found with acute udder inflammation from which streptococci were isolated that were identical with organisms isolated from the throats of several patients. Smith and Brown (16) encountered 7 epidemics of septic sore throat which occurred in Massachusetts during 1913 and 1914. Krumwiede and Valentine (17) reported an outbreak of 232 cases in June, 1914, at Rockville Center, New York. Armstrong and Parron (28) reported 42 outbreaks with 21,045 cases, occurring between 1908 and 1926, all in northern United States.

In 1918 the United States Public Health Service, through the Office of Milk Investigations, began the compilation of milk-borne epidemics of disease, including septic sore throat. During the next six years only three such epidemics are listed—43 cases at Winchester, Massachusetts, in 1920, 487 cases with 22 deaths at Portland, Oregon, in 1922, and 70 cases at Arlington, Massachusetts, in 1923. This decline in incidence corresponds with the experience of Bigelow and Forsbeck (18), who reported in the years 1907-1914 a total of 2,512 cases of septice sore throat in Massachusetts traced to milk infection, while in the years 1924-1926 there were none (table 39).

The danger from milk-borne epidemics of septic sore throat has not been eliminated, however, for during the years 1925 and 1926

Table 39.

SEPTIC SORE THROAT IN MASSACHUSETTS
(Bigelow and Forsbeck)

Year	Total cases reported	Cases traced to milk	Percent traced to milk
1907–1914	——	2,512	——
1915–1918	1401	867	61.88
1919–1923	827	118	14.23
1924–1926	415	0	0

there occurred twelve such outbreaks in the United States (table 41). The extensive epidemic in Lee, Massachusetts, with 1,000 cases and 40 deaths in a population of 4,050 people, in the spring of 1928, has still further emphasized the necessity for precaution in the production of market milk.

The Disease in Man

In the study of the Boston epidemic of septic sore throat Winslow reported that "the disease is a variable one, and single cases may present hardly a feature in common with each other, but any series of twenty cases shows at once its characteristic features."

The incubation period is short, about two days. The onset of attack is rapid, often accompanied by severe headache and acute "grippy" pains. The temperature is high, reaching 103° F. to 105° F. The throat is variable. At first there is a diffuse redness extending over the tonsils, simulating scarlet fever. Later, small isolated patches of white appear, resembling ordinary follicular tonsilitis. Still later many cases may show an extensive membrane—like exudate similar to that in diphtheria.

The first stage of the disease lasts 3 to 5 days in which there is great prostration. The patient entirely recovers in a short time provided there are no complications. Numerous cases, however, show such complications as abscesses in the peritonsilar region and cervical glands. Very severe cases may result in rheumatism, pneumonia, nephritis, or pericarditis. Death never follows the original infection but may be the result of a complication.

In the 1928 epidemic at Lee, Massachusetts, the cases were distributed among the different age groups as shown in table 40.

Table 40

EPIDEMIC OF SEPTIC SORE THROAT AT LEE, MASS.

Showing age groups affected.

Age	*1,000 Cases*	*40 Deaths*
0-15	20 per cent	3 per cent
16-29	28 per cent	3 per cent
30-44	22 per cent	3 per cent
45-59	19 per cent	31 per cent
60 and over	11 per cent	60 per cent

A further study of that epidemic indicated that 45 per cent of sick families had 1 case, 28 per cent had 2 cases, and 27 per cent had 3 cases.

Table 41.

EPIDEMICS OF MILK-BORNE SEPTIC SORE THROAT TABULATED BY OFFICE OF MILK INVESTIGATIONS
U. S. Public Health Service, 1918–1927

Year	Place	Cases	Deaths	Milk raw or pasteurized
1920	Winchester, Mass.	43	0	raw
1922	Portland, Ore.	487	22	"
1923	Arlington, Mass.	70	0	"
1924	Danbury, Conn.	89	–	"
1925	Nashua, N. H.	42	0	"
1925	Summit and Chatham, N.J.	100	2	"
1925	East Hampton, Conn.	45	0	"
1925	Logan, Ohio	332	1	"
1925	Chester, Pa.	400	0	"
1925	Mercer, Pa.	53	2	"
1926	Pleasanton, Cal.	120	2	"
1926	Guilford, Conn.	223	5	"
1926	Hamden, Conn.	95	0	certified
1926	Brunswick, Ga.	900	0	"
1926	Nashua, N. H.	100	0	raw
1926	Madison, Wis.	80	0	"

PREVENTION

The prevention of milk-borne outbreaks of septic sore throat depends upon two factors:

1. Rigid precaution in the prevention of udder infection of cattle by human cases of sore throat.
2. Proper pasteurization of all milk supplies.

Since udder infection with *Streptococcus epidemicus* often produces no apparent evidence of its presence, cases of mammitis often go unrecognized till a considerable amount of damage has been done. Even in certified dairies with regular examination of both employees and cattle, Frost, Gumm, and Thomas found by laboratory examination four cows infected with *Streptococcus epidemicus*. Ordinary market milk, therefore, especially requires careful pasteurization as a supplementary protection.

Pasteurization as a means of destroying milk streptococci has been much studied by numerous investigators. It would appear that the bovine species are so heat resistant that some of them will withstand 143° F. for sixty to ninety minutes and can subsequently be demonstrated in small numbers in the milk. The human types on the other hand are quite susceptible to pasteurizing temperatures and are entirely destroyed within the allotted time.

Park (27) collected 100 strains of streptococci from cases of septic sore throat, tonsilitis, scarlet fever, mastoiditis, glandular fever, erysipelas, and other human sources, finding every one of them killed after an exposure of 10 minutes at 140° F. or 30 minutes at 138° F. The destruction of many of the organisms was surprisingly rapid, some showing no growth after 5 minutes at 138° F.

ITEMS OF NOTE

1. Septic sore throat in man is caused by *Streptococcus epidemicus.*
2. The organism may find lodgement in the milk ducts of the cow without necessarily producing any pathological lesion.
3. The ordinary streptococci of bovine origin do not cause sore throat in man.
4. Enormous numbers of streptococci may be eliminated from the infected udder of a cow, causing an acute outbreak of sore throat among persons drinking the milk.
5. Proper pasteurization of all milk supplies is the only sure safeguard against such epidemics.

BIBLIOGRAPHY

1. REPORT OF MEDICAL OFFICER TO LOCAL GOVT. BOARD OF GREAT BRITAIN, 1875, *7,* 80.
2. BRIT. M. J., 1881, *1,* 657.
3. WINSLOW, C.-E. A.
   Jour. Inf. Dis., 1912, *10,* 73-112.
4. NOCARD and MOLLERAU
   Archiv. Vet., 1884.
5. MOORE
   Bur. of Animal Industry, U. S. Dept. Agri., 1893, Bull. 3.
6. EASTES, G. L.
   Brit. Med. Jour, 1899, *2,* 1341.
7. SAVAGE, W. G.
   Jour. Comp. Path., *22,* 361.
8. CARPENTER, C. M.
   Jour. Am. Vet. Med. Asso., 1925, *67,* 317-323.
9. HARDENBERGH, J. G., and SCHLOTTHAUER, C. F.
   Jour. Inf. Dis., 1927, *40,* 667-672.
10. DAVIS, D. J., and CAPPS, J. A.
    Jour. Inf. Dis., 1914, *15,* 135.
11. SWITHINBANK, H., and NEWMAN, G.
    Bacteriology of Milk, 1903. E. P. Dutton and Co., New York.

12. Capps, J. A., and Miller, J. L.
Jour. Am. Med. Asso., 1912, *58,* 1111-1148.

13. Mann, T. A.
Jour. Inf. Dis., 1913, *12,* 481.

14. Frost, W. H.
Public Health Reports, 1912, *27,* 1889.

15. North, C. E., White, B., and Avery, O. T.
Jour. Inf. Dis., 1914, *14,* 124.

16. Smith, T., and Brown, J. T.
Jour. Med. Res., 1915, *31,* 455.

17. Krumwiede, C., and Valentine, E.
Jour. Med. Res., 1915, *33,* 231.

18. Bigelow, G. H., and Forsbeck, F. C.
Am. Jour. Pub. Health, 1927, *17,* 1019.

19. Frost, W. D., and Bachmann, F. M.
Am. Jour. Pub. Health, 1923, *13,* 300.

20. Davis, D. J.
Jour. Inf. Dis., 1916, *19,* 236.

21. Mathers, G.
Jour. Inf. Dis., 1916, *19,* 222.

22. Frost, W. D., Gumm, M., and Thomas, R. C.
Jour. Inf. Dis., 1927, *40,* 698-705.

23. Bundesen, H.
Jour. Am. Med. Asso., 1925, *85,* 1285.

24. Brown, J. H., Frost, W. D., and Shaw, M.
Jour. Inf. Dis., 1926, *38,* 381-388.

25. Bergey, D. H.
Manual of Determinative Bacteriology. Williams and Wilkins, Baltimore, 1925.

26. Frost, W. D., and Carr, A. M.
Am. Jour. Pub. Health, 1927, *17,* 139-141.

27. Park, W. H.
Am. Jour. of Pub. Health, 1928, *18,* 710-714.

28. Armstrong, C., and Parron, T.
Public Health Reports, 1927, Supplement 62.

CHAPTER XXI

DIPHTHERIA

Diphtheria is primarily an infectious disease of man caused by *Corynebacterium diphtheriae.* On rare occasions animals may become infected, thus acting as a source of infection for man.

There have been reported in the past a considerable number of instances of infection of dogs, cats, and fowls. Bacteriological evidence, however, is either entirely lacking or is faulty, with diphtheroid organisms often mistaken for true diphtheria bacilli.

Cats have been found by several investigators to be susceptible to diphtheria toxin as well as to the bacilli. When injected subcutaneously either with toxin or with live cultures such animals exhibit definite symptoms and often die. (Klein (1, 2), Brodie (3), Yabe (4).)

Klein (5) rubbed cultures into the cornea of cats as well as injected intratracheally with definite results. Infection by feeding the organisms in milk was unsuccessful, however. Intratracheal inoculations were successfully made by Welch and Abbott (6) with the production of membranes from which the organisms could be recovered. Henke was unsuccessful in the production of membranes.

Savage (7) sought to determine experimentally whether cats were capable of harboring diphtheria bacilli. The implantation of large numbers of virulent organisms into the nasal cavities were ineffective in setting up any general or local lesion. The same was true when massive doses were applied to the throat by swabs. The organisms survived but a short period of time, usually disappearing within twenty-four hours. Savage believes that the mucous membranes of cats are unfavorable for the growth or persistence of the diphtheria bacillus, and that cats do not serve as carriers of diphtheritic infection. He analyzes the epidemiological evidence of previous cases reported, concluding that the view that cats can acquire the disease naturally is entirely without foundation.

Simmons (8), on the other hand, found two cats harboring true diphtheria bacilli, one in the nasal fossa and the other in lesions of the vocal cords. The first animal developed a croupy cough, was unable to swallow, and cried continually. About a week after the cat developed this condition, a woman with whom the cat slept, developed a typical case of diphtheria which proved fatal. The organisms isolated from both the woman and the cat were virulent for guinea pigs as proved by the injection of two animals, one of which was protected by antitoxin.

Domestic fowls were found by Litterer (9) capable of not only harboring diphtheria bacilli, but of transmitting the disease to man. Two hundred and fifty-six human cases suffering from acute or carrier diphtheritic infection were investgiated, where the family owned fowls suffering from croup, fowl diphtheria, cholera, etc. A total of 1,126 sick fowls were examined; in two flocks several fowls were found harboring virulent diphtheria bacilli as proved by morphological and virulence tests. In each instance a child in the family owning the fowls showed similar virulent organisms. Successful inoculations of the virulent diphtheria organisms isolated from both the fowls and the children were made into other fowls suffering from croup; later the infection in the experimental fowls was cured with diphtheria antitoxin.

Cows are quite susceptible to diphtheria toxin. They have been recommended from time to time as a substitute for the horse in the production of antitoxin, but they are not altogether satisfactory. Diphtheria bacilli, when injected under the skin, were found by Klein (2) to kill cows in as short a period as eleven days. Fifteen animals so treated either died or showed marked reactions. Wladimiroff had a similar experience but Abbott (10) could not confirm Klein's findings on two cows with which he worked.

Cows fed diphtheria bacilli by mouth exhibited no ill effects (Klein). Diphtheria bacilli injected into the udder through the teat by Wladimiroff killed a cow in ten days, although diphtheria toxin administered in the same way caused no trouble.

Dean and Todd in 1902 (11) found two cows with scabby teats from which diphtheria bacilli were isolated. The animals were implicated in a diphtheria epidemic. The authors concluded that the cows were infected by the milker. Ashby in 1906 (12) similarly found diphtheria bacilli in ulcers on the teats of a cow implicated in an epidemic. The milker of the cow, however, was probably responsible for this condition since he lived in the house where the first case of the epidemic originated.

Henry (13) describes an epidemic of 32 cases where a cow was involved. A girl who did the milking had a lesion on the finger which proved to be harboring diphtheria bacilli. With her removal the epidemic ceased, only to flare up again two weeks later. It was found that one of the cows had developed a sore on its teat which harbored diphtheria bacilli. The organisms isolated were virulent by the usual protection tests.

Graham and Golaz (14) reported a cow with sores on the teats, covered with thick, black scabs. When the scabs were removed a ragged, ulcerated surface was left that exuded a mucopurulent fluid. The teats were tender and the sores did not heal. Cultures showed virulent diphtheria bacilli present. An investigation revealed that the cow had been infected by a milker who was a diphtheria carrier. This cow was responsible for fifty-two cases of diphtheria, mostly among adults.

Since diphtheria bacilli localize at the point of entry and produce a toxemia rather than a bacteremia, such organisms do not find their way into the milk of infected cows unless the primary lesions are on the teats or udder. Instances of cutaneous diphtheria on the teats or udder of the cow, as cited above, may be even more numerous than reported. Cases of true mastitis or udder infection with the diphtheria bacillus have not been reported and it is not known whether they occur under natural conditions or not.

Numerous epidemics of diphtheria spread by cow's milk have been reported. In 1908 Trask (15) collected 51 such records which had occurred up to that time and Armstrong and Parron (18) added 25 more from 1908 to 1926 in the United States. In the latter study pasteurized milk was implicated once, certified milk once, ice cream once, and butter once. The epidemics were mostly in the northern states, although one epidemic occurred at Austin, Texas, and one at Charlottesville, Virginia.

Recently the number of milk-borne diphtheria epidemics has fallen off due to the increase of pasteurization. From 1918 to 1923 the United States Public Health Service found by a questionnaire, records of only two such epidemics. Milk in such instances has been commonly thought to receive its infection with virulent diphtheria bacilli directly from a human carrier or case either on the farm where the milk is produced or en route to the consumer.

Horses are quite susceptible to diphtheria toxin when injected for the purpose of producing antitoxin, and consequently considerable care must be used on initial injections. True diphtheria occurs but rarely, however. Cobbett (16) reports such a case in a pony infected by its rider. Minett (17) likewise recorded several cases of ulcerative lymphangitis in horses infected with diphtheria bacilli probably from human cases.

Other animals exhibit some susceptibility to toxin but apparently do not contract the disease naturally. Sheep have been used to some extent for the production of antitoxin and may succumb from overdoses of toxin as noted by Roux and Yersin in 1889. Wernicke in 1893 likewise killed a sheep by gradually increasing the doses of diphtheria culture. Goats have recently come into use for the production of diphtheria antitoxin to be used in toxin-antitoxin mixtures. Ledingham found that goats would succumb to large injections of toxin (250 cc.). Wladimiroff injected diphtheria cultures into the teats of three lactating goats. Only one of them exhibited any constitutional symptoms. Diphtheria bacilli were found in the milk for a period of six days, but they rapidly deminished in numbers. Dogs have been utilized by various workers in experimental investigations. They are susceptible to injection of both toxins and cultures. Unconfirmed reports of natural diphtheria infection in dogs have been recorded. Brandt in 1908 obtained from a dog a culture which he considered true diphtheria, but it did not kill guinea pigs when injected. I have had cases reported to me of diphtheria infection in dogs, but complete laboratory confirmation was lacking.

The laboratory animals vary in susceptibility. Rats and mice are quite immune. Guinea pigs are very constant in susceptibility and hence are the animals of choice in experimental work. The standardization of both diphtheria toxin and diphtheria antitoxin is based upon the amount of toxin required to kill a guinea pig weighing 250 grams in four days. Rabbits were originally thought to be less susceptible than guinea pigs but a comparative study made by Goodman in 1907 indicated that rabbits exhibited very small resistance.

Monkeys were found by Burnet in 1910 rather resistant to infection. Seventeen experiments were made by rubbing virulent cultures into the abraded mucous membrane of young animals. Eleven experiments were entirely negative, in five the results were slight, while in one animal which was ill at the time of inoculation, the lesions were extensive.

Eels were shown susceptible to diphtheria toxin by Pettit on two occasions, while alligators were found highly susceptible by Metchnikoff. Frogs, when injected with toxin, remained well at low temperatures, but developed paralysis when maintained at 37° C.

Items of Note

1. Diphtheria is primarily an infection of man.
2. On rare occasions animals may become naturally infected from human sources.
3. Most of the reports in the literature of animal infection are unconfirmed by complete laboratory diagnosis.
4. The cat is a doubtful menace; chickens have been found harboring virulent diphtheria bacilli; horses may contract the infection.
5. The cow may become infected upon the udder and thus be a serious menace by way of the milk supply.

BIBLIOGRAPHY

1. Klein, E.
Rep. Med. Off. Local Gov. Bd., London, 1888, *18,* 425.
2. Klein, E.
Rep. Med. Off. Local Gov. Bd., London, 1889, *19,* 143-214.
3. Brodie, T. G.
Jour. Path. and Bact., 1897, *4,* 460.
4. Yabe, S.
Jour. Pharmacol. and Exp. Ther., 1922-23, *19,* 1.
5. Klein, E.
Rep. Med. Off. Loc. Gov. Bd., London, 1890, *20,* 219-241.
6. Welch, W. H., and Abbott, A. C.
Johns Hopkins Hosp. Bull., 1891, *2,* 25.
7. Savage, W. G.
Jour. Hyg., 1919-20, *18,* 448.
8. Simmons, J. S.
Am. Jour. Med. Sci., 1920, *160,* 589.
9. Litterer, W.
South. Med. Jour., 1925, *18,* 577-581.
10. Abbott, A. C.
Jour. Path. and Bact., 1894, *2,* 35.
11. Dean, G., and Todd, C.
Jour. Hyg., 1902, *2,* 194.

12. Ashby, A.
Pub. Health, 1906, *19,* 145.
13. Henry, J. E.
Jour. Am. Med. Asso., 1920, *75,* 1715.
14. Graham, M., and Golaz, B. S.
Jour. Am. Med. Asso., 1922, *79,* 1300-1301.
15. Trask, J. W.
Hygienic Lab. Bull., No. 41, 1908, 21-147.
16. Cobbett, L.
Centralbl. f. Bakt., 1900, I Abt., *28,* 631.
17. Minett, F. C.
Jour. Comp. Path. and Thera., 1920, *33,* 267.
18. Armstrong, C., and Parron, T.
Public Health Reports, 1927, Supplement 62.

CHAPTER XXII

SCARLET FEVER

Scarlet fever is primarily an infectious disease of man caused by *Streptococcus scarlatinae.* Cows may become infected from human sources and pass the disease back to man through the milk.

Many outbreaks of scarlet fever have been attributed to contaminated milk supplies in the past. Trask (8) listed 125 such epidemics which occurred prior to 1908. Armstrong and Parron (14) added 40 more outbreaks in the United States from 1908 to 1926. All occurred in the northern states, southwestern Ohio being the most southern locality.

Evidence of contamination of the milk supply with scarlet fever organisms has, until very recently, been based upon epidemiological studies. In 1923 the Dicks, as well as Dochez, differentiated the scarlet fever streptococcus from other organisms in the group, making bacteriological evidence possible. Previous to that time a case of scarlet fever on the farm or in a dairyman who handled the milk was considered sufficient evidence upon which to base responsibility for an epidemic.

The responsibility of the cow was suggested from time to time in the absence of other evidence. Power (1) in 1882 attributed an outbreak to contamination by a cow suffering with puerperal fever. Klein at the same time showed that cows inoculated subcutaneously with material from a human case of scarlet fever developed an abscess at the site of inoculation and material from such an abscess could produce a similar lesion when injected into another cow.

In 1885 Power (2), Cameron (3), and Klein (4) encountered another epidemic which they attributed to bovine origin, lacking any human sources. A vesicular eruption on the udder and buttocks of several animals accompanied by constitutional symptoms and discharge from the eyes and nose, with sore throat (Hendon disease) was thought to be the cause. Russell (5) in 1888, Hill (6) in 1890, and Jones (7) in 1909 all reported similar instances where a milk epidemic of scarlet fever occurred and cows in the responsible herd were found suffering from a condition similar to that described by Power, Klein, and Cameron. Bacteriological evidence was, of course, entirely lacking.

Busey and Kober (8), in 74 epidemics included in Trask's compilation, state that in 20 instances the infection was attributed to disease among the milk cows. In 4 cases the puerperal condition of the cow is blamed, in 9 there was disease of the udder or teats, in 6 there was loss of hair and casting of the skin, while one cow was suffering from a febrile condition.

Jones and Little (9) were the first to furnish bacteriological evidence that cows could be infected with scarlet fever streptococci. In a scarlet fever outbreak of 200 cases studied by the New Jersey State Department of Health, the usual relation of a case of scarlet fever on one of the farms supplying the milk was found. More careful observations indicated that one of the cows was suffering from an acute injury of the teat. An investigation by Jones and Little of the milk from this cow indicated that as many as 345,000,000 streptococci per cubic centimeter were being eliminated. The organisms answered all the necessary requirements of scarlet fever streptococci of human origin (10).

Injection of these organisms into one of the other teats of the same cow produced no abnormality nor could streptococci be found in the milk from that quarter, until still another quarter was injected when both of these showed streptococci which gradually diminished in two weeks.

A normal cow was injected by means of a teat tube with 15 to 20 of the scarlet fever streptococci isolated from the first cow. There was a severe general reaction beginning within two days, with severe fever and reddening of the skin. The udder was swollen and the milk purulent. On the second day 1,200,000,000 streptococci per cubic centimenter were eliminated. The injection of another teat of this cow resulted in a condition similar to the second injection of the other cow, in that the quarter showed no abnormalities but excreted for a time a comparatively small number of organisms (556,000 per cc. at the most). This would indicate a lack of immunity of the other quarters when infection existed in one quarter.

Another epidemic of scarlet fever studied by the Connecticut State Department of Health resulted in the discovery of a cow harboring typical scarlet fever streptococci of human origin (9).

Jones (11) showed that the usual view that milk-borne epidemics of scarlet fever are due to a human case or carrier

Table 42

EPIDEMICS OF MILK-BORNE SCARLET FEVER

Tabulated by Office of Milk Investigation, 1918–1927.

Year	Place	Cases	Deaths	Milk raw or pasteurized
1919	Greenwich, Conn.	18	0	raw
1919	Orange, N. J.	10	0	“
1920	Providence, R. I.	31	0	past.
1921	Ossining, N. Y.	24	0	—
1922	Croton, N. Y.	44	1	—
1922	Huntington, N. Y.	?	?	—
1922	Jamestown, N. Y.	25	–	—
1922	Beloit, Wis.	17	1	raw
1923	Winchestertown, Mass.	7	0	“
1923	Kewanee, Ill.	23	1	“
1923	Madison, Wis.	33	0	“
1923	Buffalo, N. Y.	57	3	past.
1923	Pittsfied, Mass.	8	0	raw
1923	San Bernadino, Cal.	208	2	“
1924	Helena, Mont.	38	0	“
1924	Pittsfield, Mass.	7	0	“
1924	Bristol, Conn.	133	0	“
1924	Cohoes, N. Y.	82	–	“
1924	Pontiac, Mich.	5	0	“
1925	Helena, Mont.	11	1	“
1925	Binghamptom, N. Y.	25	0	“
1925	Binghamptom, N. Y.	19	0	“
1925	Scio, Ohio.	81	2	“
1926	Clinton, Mass.	80	0	“
1926	St. Johns, Mich.	59	0	“
1926	Lakefield, Minn.	67	0	“
1926	Deposit Village, N. Y.	51	2	“
1926	Troy, N. Y.	14	1	“
1927	Washington, N. J.	199	0	“
1927	Riverside & Paramus, N. J.	9	–	“
1927	Schuylerville, N. Y.	29	0	“
1927	Kalispell, Mont.	139	3	“
1927	Janesville, Wis.	22	2	“

on the farm or in the dairy is erroneous. Milk is not only an inhibitive agent in which such organisms fail to grow, but it contains an active principal which greatly diminishes the number of streptococci at room temperature, with a slower diminution at refrigerator temperature.

Jones concludes that "the opinion that severe outbreaks of scarlet fever result from human contamination of milk must be viewed with considerable doubt." It is possible that a few individuals may contract the disease through direct human contamination of the milk, but the occurrence of epidemics would imply a much heavier inoculation of the milk than would be probable by this means. The enormous numbers of streptococci shed from the udder of a single cow infected with this organism may contaminate a large bulk of milk and even after some of the streptococci have perished, still show sufficient numbers of living organisms to infect man. This is a more reasonable explanation of milk-borne epidemics.

Animals other than the cow are probably of small importance in the spread of scarlet fever. Reports have been made from time to time concerning infection of dogs in contact with human cases, but all lack bacteriological confirmation. Numerous attempts were made before the discovery of *Streptococcus scarlatinae* to induce infection in laboratory animals with material direct from human cases, but all were unsuccessful. Several workers were able to produce lesions in monkeys and chimpanzees with human material while others failed. The failure of Hektoen and Weaver in producing the disease in monkeys fed on milk to which had been added material from the throats of scarlet fever patients, is explained by the work of Jones cited above.

Since 1923 experimental infection has indicated the susceptibility of certain animals. The dog is highly susceptible to the infection of a living culture of the organism, succumbing in 3 to 5 days from an acute hemorrhagic nephritis. Killed cultures and culture lysates are also highly toxic for dogs (12). Goats are quite susceptible to scarlet fever toxin and have been used to a certain extent as a substitute for human subjects in standardizing such toxin. Mice, rats, guinea pigs, cats, chickens, pigeons, monkeys, calves, and sheep have been found entirely unsusceptible to toxin while rabbits and pigs give conflicting results (13).

The prevention of scarlet fever of animal origin requires attention to several details. First no human case of scarlet fever must have contact with lactating cows. Even a few organisms will be sufficient to infect the teats. In the presence of a suspected milk-borne epidemic of scarlet fever careful examination must be made of all cows in the herd for possible infection. Finally pasteurization of the milk will be a complete safeguard, for scarlet fever streptococci are easily killed by heat at 140° F. for 30 minutes.

Items of Note

1. Scarlet fever is primarily a human infection.
2. Cows may become infected in the udder, shedding enormous numbers of scarlet fever streptococci into the milk.
3. Milk, under ordinary conditions, prevents the growth of scarlet fever streptococci, even killing them to a certain extent.
4. Prevention of milk-borne scarlet fever requires first that cows be kept free from contact with human cases of scarlet fever and second, proper pasteurization of all milk.
5. Animals other than the cow are probably of little importance in the spread of scarlet fever.

BIBLIOGRAPHY

1. Power, W. H.
Rep. Med. Off. Local Gov. Bd., London, 1882-83, *12,* 63.
2. Power, W. H.
Rep. Med. Off. Local Gov. Bd., London, 1885-86, *15,* 73.
3. Cameron, J.
Rep. Med. Off. Local Gov. Bd., London, 1885-86, *15,* 85.
4. Klein, E. E.
Rep. Med. Off. Local Gov. Bd., London, 1885-86, *15,* 90.
5. Russell, J. B.
Sanitary Jour., 1888-89, *12,* 70-74.
6. Hill, B.
Pub. Health, 1890-91, *3,* 487.
7. Hamar, W. H., and Jones, T. H.
Jour. Comp. Path. and Therap., 1909, *22,* 363.
8. Trask, J. W.
Hygienic Lab. Bull. No. 41, 1908, 21-147.
9. Jones, F. S. and Little, R. B.
Jour. Exp. Med., 1928, *47,* 945-956.
10. Jones, F. S., and Little, R. B.
Jour. Exp. Med., 1928, *47,* 957-964.
11. Jones, S. F.
Jour. Exp. Med., 1928, *47,* 965-976.
12. Duvall, C. W., and Hibbard, R. J.
Jour. Exp. Med., 1927, *46,* 379-390.
13. Kirkbride, Mary B., and Wheeler, Mary W.
Jour. Imm., 1926, *11,* 477-497.
14. Armstrong, C., and Parron, T.
Public Health Reports, 1927, Supplement 62.

CHAPTER XXIII

OTHER HUMAN DISEASES SOMETIMES CONTRACTED BY ANIMALS

Tuberculosis of human origin is rare in most animals. The cow, horse, dog, goat, cat, and rabbit are difficult to infect artificially with this type of organism even when injected under the skin, and spontaneous infection is very rare or unknown. Guinea pigs are easily infected by injection but spontaneous infection with the human type of organism is rare and no known cases in man have been attributed to these animals. Anthropoid apes and monkeys are even more susceptible than man to the human tubercle bacillus. Close association with such an infected animal might be dangerous to a person. Parrots and cockatoos may contract the human type of tuberculosis and spread the organisms to persons associated with them through dust from the cage. Hogs are as susceptible to the human as to the avian or bovine type but less often contract it spontaneously. Butler and Marsh, however, found 30 per cent of the hogs fed on garbage from a tuberculosis sanitarium to be infected with the human type of organism. Since the life of a hog is usually short, such infection does not develop beyond the glandular stage. Old hogs may show lesions of the lung and liver and the living animals spread enough organisms to be a menace to man. Workers in slaughter houses are exposed to infection from hogs, while the meat of infected hogs in some cases is capable of conveying infection if eaten without proper cooking. (See chapter I.)

Smallpox is a disease of man which cattle may contract, but in a very modified form. This was the belief of Jenner and has been substantiated by experimental evidence by several investigators. When the infection has passed through the cow, the virus will never again produce the symptoms of smallpox in man. It will bring about a mild form of infection, however, which protects the individual against subsequent smallpox. The administration of vaccine virus, therefore, has come to be the principal factor in the fight against smallpox. It sometimes happens that a recently vaccinated individual will carry the infection of cowpox back to the cow, thus starting an epidemic among cattle. Susceptible persons may vaccinate themselves accidentally in caring for or milking such infected cattle. (See chapter VII.)

SECTION FOUR

ANIMALS AS PASSIVE CARRIERS OF DISEASE ORGANISMS

SECTION FOUR

ANIMALS AS PASSIVE CARRIERS OF DISEASE ORGANISMS

CHAPTER XXIV

BOTULISM

Botulism is caused by the toxin of *Clostridium botulinum* in food materials. The disease may affect either man or animal; man is not usually infected directly from an animal case, but animals and birds have been found to be responsible to a certain extent for the spread of the organisms.

Botulism has been known since the latter part of the eighteenth century. In Germany, especially, numerous outbreaks were traced to preserved meats (hence the name from "botulus," a sausage).

In recent years other foods than meat and sausages have usually been responsible for the trouble, so that the name has lost its original significance.

The botulinus bacillus was discovered by Ermengen (6) in 1897 in ham preserved in brine. The organism itself is an obligate anearabe but in symbiosis with other organisms it may grow under aerobic conditions. It forms spores which are quite resistant to unfavorable conditions, requiring heat at boiling temperature for several hours. Three types of the botulinus bacillus have been isolated:

- Type A—cause of the majority of American outbreaks. Found in canned ripe olives, spinach, corn, etc. Also the cause of forage poisoning in animals and limberneck in poultry.
- Type B—cause of the majority of European outbreaks, but found in America in several outbreaks. Spores in nature quite common in America, however.
- Type C—cause of limberneck in chickens but has never been found in human outbreaks.

The spores of the botulinus bacillus are widely distributed in nature and probably multiply in the soil in symbiosis with other organisms. They are present also in the intestines of certain normal animals, which probably act to a certain extent as endemic foci. Meyer and Geiger (5) report an instance where the owner of a small vegetable garden for ten years grew string beans and canned them at home without any spoilage. When the garden was fertilized with manure, however, the contents of the jars spoiled and the botulinus bacillus was demonstrated. Dickson and Burke (2) isolated the organism from the feces of a hog, and Easton and Meyer from the feces of three hogs and two cattle. Forty-five other specimens from cattle, sheep, horses, and chickens were negative, however, and it was concluded that animal excreta contribute but little to the pollution of the soil with the botulinus bacillus.

Birds act as passive carriers of infection. Dickson and Burke found botulinus organisms on bird pecked cherries and Meyer and Geiger found them on bird pecked apricots, cherries, and olives.

Man does not act as a carrier of botulinus spores. Easton and Meyer examined the stools of eight-eight healthy persons with negative results, although it was known that spores were being ingested on raw fruits and vegetables served as food.

Botulism in animals has been reported from time to time (4). In poultry two outbreaks occurred in 1910 with 103 outbreaks during the next ten years. Fifty-six of these occurred in California and 31 in the State of Washington, with the others in Colorado 2, District of Columbia 1, Illinois 1, Indiana 1, Michigan 1, New Jersey 1, Nevada 5, and Oregon 4. In 22 instances the outbreaks in chickens were caused by careless disposition of food which had caused human outbreaks and to which the chickens had access.

Botulism in horses is a serious affliction in some localities.

It is estimated that 3,000 horses perished from so-called "forage poisoning" in the five Western states of California, Colorado, Oregon, Nevada, and Montana during the five-year period of 1915 to 1920. Moldy and spoiled hay, potatoes, corn, and other feeds containing botulinus toxins were the responsible agents. Sometimes, however, the feed appeared wholly normal and yet was highly poisonous.

Cattle are rather infrequently infected, although outbreaks have been reported by Graham and Schwarze and others. Hogs show a marked resistance to botulinus toxin. Goats are very susceptible, while dogs may develop the disease on occasion but are usually resistant. Cats, rabbits, guinea pigs, and mice are extremely susceptible to subcutaneous injections of the toxins; white rats, frogs, and fishes are almost completely resistant.

Botulism in man was first observed in the United States in 1915 by Nevin, who isolated the botulinus bacillus from cream cheese. Dickson, however, was able to trace records of the disease back to 1899. From that time to 1928 there occurred in the United States and Canada, with one outbreak in England and one in Argentina, 156 outbreaks, or a total of 529 cases with 357 deaths (7). The foods involved were mostly string beans, cottage cheese, corn, peas, spinach, and ripe olives.

Prevention and Control

The prevention of botulism depends upon careful methods of canning and preserving food and thorough cooking just before the food is eaten. Toxin is quickly destroyed by heat, but it may reform in twenty-four hours and require reheating. Commercially, botulism is no longer a problem, the last case from commercially canned products having occured in 1925. Education in the home is still necessary, however.

Antitoxin for the cure of the disease has been prepared against both types A and B. Antitoxin of one type will not protect against the other type, however. The serum is of little avail after symptoms begin.

Items of Note

1. Botulism is caused by the toxin of the *botulismus bacillus* formed in food.
2. The organism free from toxin is harmless to man and animals by ingestion.
3. The toxin is quickly destroyed by heat at boiling temperature.
4. Botulinus organisms are present in the intestinal tract of certain healthy animals.
5. The organisms are found widely spread in nature where they probably multiply.
6. Prevention consists in careful preparation of preserved foods and boiling the product just before it is eaten.

BIBLIOGRAPHY

1. Bengston, Ida
U. S. Public Health Service, Hygienic Lab. Bull., 136, 1-96. March, 1924.

2. Dickson, Ernest C.
Rockefeller Inst. for Med. Res., Monograph 8, 1-117, 1918.
3. Easton, E. J., and Meyer, K. F.
Jour. Inf. Dis., 1924, *35*, 207-212.
4. Geiger, J. C., Dickson, E. C., and Meyer, K. F.
U. S. Public Health Service, Public Health Bull., 127, 1-119, March, 1924
5. Meyer, K. F., and Geiger, J. C.
Public Health Reports, 1921, *36*, 4-6.
6. Von Ermengen, E.
Zeitschr. f. Hyg., 1897, *26*, 1-56.
7. Anon.
Jour. Am. Med. Asso., 1928, *90*, 764.

CHAPTER XXV

TETANUS

Tetanus is the result of wound infection with the tetanus organism. The disease affects all mammals, both man and animal, in varying degrees of severity. The natural habitat of the organism is the intestine of the herbivorous animals from which it is spread universally. To a less degree man likewise acts as an intestinal carrier.

Tetanus has been recognized since the time of Hippocrates. It has always been a scourge accompanying war. In 1885 Nicolaier demonstrated the presence of the infecting organism in garden earth and in 1889 Kitasato isolated it in pure culture.

Clostridium tetani is an anaerobic spore bearing organism of considerable resistance. The spores are destroyed by direct sunlight but when protected will remain alive and virulent for years. They are not susceptible to the ordinary disinfectants or to boiling water except when exposed over long periods.

The distribution of the spores is universal. They are present in the soil practically everywhere, but are especially abundant in soil fertilized with human or animal excreta.

Animals may carry tetanus organisms in their intestines over long periods of time without harm to themselves. Noble (1) found 18 per cent of the horses in the vicinity of New Haven so affected. The presence of the organisms in the intestine is not always due to constant reinfection for Noble found one horse to harbor the organism continuously for four months, although other horses in the same stable were free. Apparently the organisms grow and multiply in the intestine and are eliminated in great numbers with the dejecta.

According to Park, 15 per cent of the horses and calves in the vicinity of New York City harbor tetanus bacilli in their intestines. Ninni (3) cultivated the organisms from eight different portions of the stomach and intestine in forty-two rabbits and guinea pigs with very constant results.

Man may carry the organisms in his intestine, likewise. Pizzini (4) found 5 per cent of human feces to harbor tetanus bacilli. Men working around horses and stables, however, were carriers in about 30 per cent of the instances, while men in other occupations showed about 2.2 per cent. Tulloch (5) studied the feces of 21 men returned from overseas army service in 1919, obtaining positive results in 33 per cent; from 33 civilians in England 16 per cent were positive. Ten Broeck and Bauer (6) in 1921 examined the stools of 78 Chinese in Peking and found 34.7 per cent harboring tetanus bacilli; in 1924 they examined 539 more such specimens from various parts of China, finding 26.5 per cent positive. Buzello and Rahmel (7) in Germany found 20 per cent of the feces from 40 individuals positive. Van der Reis (8) found tetanus bacilli in practically pure culture in the large intestine of one of his patients. Bauer and Meyer (9) studied 487 specimens of human dejecta in California of which 24.6 per cent were positive. In addition 43 specimens from 19 other states were examined, of which 9 showed tetanus bacilli. Sex, age, and occupation played no part in the distribution of carriers. No atoxic strains were found in the 129 cultures isolated.

Although the natural habitat of the tetanus bacillus is the intestine of the herbivorous animal (the horse or the cow), spores of the organism have been found in numerous locations. McCoy showed them in hay dust, in the mortar of old masonry, in the dust from horse hair, in the dust in houses, barracks, and hospitals, and in court plaster (10). It is only natural to expect to find such a resistant organism anywhere that the wind might carry the dust of the soil.

Gelatin sold for human consumption may contain numerous spores. In Professor Rettger's laboratory in New Haven for several years the stock culture of tetanus, for the demonstration of the disease in laboratory animals, was a package of household gelatin.

Tetanus, whether in man or animals, is a wound infection requiring a broken skin for the passage of the organisms. By mouth neither the organism nor its toxin is harmful. The organism locates in the wound, where, if anaerobic conditions are present, it proceeds to grow and secrete its powerful toxin. The organism does not wander far from its portal of entry. It has been found in the lymphatics, liver, and spinal fluid (15) on a few occasions, however. The toxin invades the nerve tissue for which it has a special affinity, thus causing the characteristic symptoms.

Among domestic animals the horse is most susceptible to the disease, followed by sheep and goats in order. Cattle are only slightly susceptible, although they may develop the disease upon artificial inoculation. Dogs and cats are seldom affected. Among the laboratory animals guinea pigs and mice are extremely susceptible, but rats and rabbits require larger amounts of material. The hen is quite refractory but will succumb to very large doses.

The prevalence of the disease in man varies in different localities according to the contamination of the soil with tetanus spores. For instance the Hudson River Valley and Long Island show a much higher incidence than do other parts of the country—a fact that coincides with the contamination of the soil.

The nature of the wound influences the development of the tetanus organism. Puncture wounds such as those caused by stepping on a nail are bad. Wounds infected with other organisms, such as the Welch bacillus, favor the development of the tetanus bacillus.

Gunshot wounds, either from bullets or blank cartridges, seem to be especially dangerous. The number of cases of tetanus following Fourth of July celebrations some years ago caused the American Medical Association to initiate a campaign in the treatment and care of wounds following accidents from fireworks. The deaths fell from 406 in 1903 to zero in 1916 and have remained at a low level since then.

Tetanus in American soldiers during the World War was controlled by the administration of tetanus antitoxin to every wounded man at the dressing station on the battle front at the same time he received the first dressing of his wounds. Among many thousands of wounded whom I saw, there was only one case of tetanus, which occurred in a German prisoner who had in some manner missed his prophylactic injection of antitoxin. The rate for the American Army in 1917-18 was 1 in 6,224 wounded (36 cases of tetanus in 224,089 war wounds) against the British rate in September, 1914, before antitoxin was used, of 1 in 111.

The presence of tetanus spores on the instrument causing the wound is not necessary for infection. Blank cartridges from several manufacturers were examined by Wells with negative results. The clothing and the skin are liable to harbor tetanus spores in greater or less numbers, especially around the feet. Such organisms are carried into the wound.

Tetanus of the operating room has not been unknown, especially in operations of the abdomen. More serious however, is tetanus following child birth, a problem giving much concern to obstetricians. Tetanus of the new born—trismus neonatorum—is a very common cause of death in the tropics. Infection takes place by way of the umbilical cord.

Rare cases of tetanus occur with no apparent lesion. Several theories have been advanced for this. Spores may lie dormant in wound or scar tissue for months or years until released to proliferate and produce toxin. They may be inhaled with street dust where they enter the body through the inflamed tissue of the respiratory tract or slight lesions of the nose. They may be carried in a dormant condition to the lymph glands, liver or other parts of the body where they eventually cause the disease.

Biological products in the past have been known to carry tetanus spores and cause the disease when applied. In India in 1902 there were 19 deaths following the administration of Haffkines plague vaccine to 107 persons. The tetanus bacillus was apparently present with the plague bacillus growing in symbiosis with it and thus producing toxin.

In 1901 in St. Louis seven children died from tetanus following the administration of diphtheria antitoxin. The serum in this instance was sterile, but the horse from which the serum had been withdrawn was in the incubation stage of tetanus and thus carried in its blood stream a large amount of tetanus toxin (11).

Smallpox vaccine was occasionally contaminated with tetanus spores in the days before governmental supervision. On July 1, 1902, all vaccine virus as well as other biologics, sold in interstate traffic in the United States, was required to come from a manufacturing plant licensed by the United States Public Health Service. Elaborate precautions against, and tests for tetanus since then have been compulsory, making it impossible for material so contaminated to find its way into the market.

Anderson (12) studied all the cases of tetanus developing after vaccination between 1904 and 1915. Forty-one such instances had occurred but not once could tetanus spores be found in the virus in the same stock as that used for vaccination. A study of commercial vaccine—virus sufficient to vaccinate 200,000 people showed no indication of the presence of tetanus organisms. Among 385,000 men vaccinated in the United States Army 6 cases of tetanus developed, none which had any relation to the vaccine-virus. Among 200,000 persons vaccinated in the United States Navy two developed tetanus, neither of which cases had any relation to the vaccine-virus. The causes for such cases are several, such as uncleanliness, accidental removal of the scab, bunion pads, etc., (see Chapter VII on cowpox).

Infection of a wound with the tetanus organism, followed by growth and the production of toxin in the wound does not always result in a case of tetanus. Some strains of tetanus bacilli produce toxin of such a low grade that it has no effect upon the body. Natural immunity is exhibited by some persons due to tetanus antitoxin in the blood stream. According to Ten Broeck and Bauer (13) antitoxin in the blood stream is correlated with the tetanus organism in the digestive tract. Twenty-six persons who exhibited antitoxin likewise were intestinal carriers while 36 persons who exhibited no antitoxin were free from the organism in the intestine (with only two exceptions).

Mortality from tetanus infection depends upon the care which the case has received. Before the introduction of tetanus antitoxin, the mortality was about 85 per cent (14). In the British Army during the World War 2,152 soldiers developed the disease, of which 1,011 died—47 per cent. However, among 715 who received no antitoxin or who exhibited a very short incubation period and received antitoxin in France, the death rate was 71.2 per cent, while among 1,437 men treated with antitoxin in England the death rate was only 34.8 per cent.

The longer the incubation period the better are the chances of the patient's recovery; the sooner the patient receives antitoxin the better are his chances also.

The prevention of tetanus therefore depends upon the administration of antitoxin as soon after infliction of the wound as convenient, but in any case before the organism has had a chance to proliferate in the tissue and toxin-fixation of the nerve tissue has occurred.

ITEMS OF NOTE

1. The natural habitat of the tetanus organism is the intestine of the herbivorous animals (horse and cow).
2. Man may carry the tetanus organism in his intestine without harm to himself.
3. Tetanus organisms and tetanus toxin by mouth are harmless.
4. Tetanus is a wound disease, requiring a broken skin for entry.
5. Tetanus spores are distributed universally. They are much more abundant in highly fertilized soil.
6. Tetanus organisms do not ordinarily multiply outside the animal body.

7. The incidence of tetanus in man is correlated with the contamination of the soil with spores.
8. The cure of tetanus by use of antitoxin is not entirely satisfactory, nearly half the cases dying.
9. The prevention of tetanus by use of antitoxin immediately after infliction of a wound is quite certain.

BIBLIOGRAPHY

1. Noble, W.
   Jour. Inf. Dis., 1915, *16,* 132.
2. Park, W. H., and Williams, A. W.
   Pathogenic Microorganisms, 8th Edition, 1924, 496. Lea & Febiger, New York.
3. Ninni, C.
   Abst. Jour. Am. Med. Asso., 1921, 76, 1617.
   Annali d'Igriene, 1920, *30,* 756.
4. Pizzini
   Baumgartens Jahresbericht, 1894, *16,* 236.
5. Tulloch, W. J.
   Jour. Hyg., 1919, *18,* 103-203.
6. Ten Broeck, C., and Bauer, J. H.
   Jour. Exp. Med., 1922, *36,* 261.
7. Buzello and Rahmel
   Arch. f. klin. Chir., 1924, *80,* 660.
8. Van der Reis, V.
   Ergebn. d. inn Med. u. Kinderlk, 1925, *27,* 77.
9. Bauer, J. H., and Meyer, K. F.
   Jour. Inf. Dis., 1926, *38,* 295-305.
10. McCoy, G. W.
    Public Health Reports, 1917, *32,* 1450.
11. Bolton, Fisch, and Walden
    St. Louis Med. Rev., 1910, *44,* 361.
12. Anderson
    Jour. Am. Med. Asso., 1915, *65,* 194.
13. Ten Broeck, C., and Bauer, J. H.
    Jour. Exp. Med., 1923, *37,* 479.
14. Stone, W. J.
    Jour. Am. Med. Assoc., 1922, *78,* 1939-1943.
15. Lacy, G. R., and Murdock, C.
    Jl. Lab. and Clin. Med., 1921, *7,* 100-103.

Chapter XXVI

GAS GANGRENE

Gas gangrene in man is caused by the invasion into injured tissues of certain anaerobic spore-forming organisms belonging to the group of which the Welch bacillus is a member. These organisms are frequently found in the intestinal canal of normal animals, where they lead a saphrophytic existence. The disease is a pestilence of war, being rarely met with in peace times.

There are several organisms associated with gas gangrene. At one time it was suposed that the Welch bacillus alone was the causative agent. Studies made during the World War however, showed that other organisms, some of them non-pathogenic in themselves, were present and enhanced the pathogenicity of the Welch bacillus. Other members of the group are even more pathogenic than the Welch bacillus.

In a chart developed at the school of Wound Bacteriology, organized at Dijon, France, during the World War by the Medical Department of the United States Army, there are listed 23 organisms associated with gas gangrene.

B. perfringens
V. septique
B. sporogenes
B. putrificus
B. tertius
B. bifermentans
B. oedematiens
B. fallax
B. aerofetidus
B. histolyticus
B. bellonensis
B. du gasoedem
B. sarcemphysematodes hominis
Anaerobic streptococcus
B. v. Hibler VI
B. v. Hibler VII
B. v. Hibler X
B. v. Hibler XI
B. anthracoides
L. Coccobacillus verdunensis
B. pseudoedematis maligni
B. septique aerobie
B. aerogenes aerophilis agilis

Weinberg and Sequin (1) showed the relative frequency of infection with the Welch bacillus group in war wounds (table 43).

Clostridium welchii (Bacillus aerogenes capsulatus, Bacillus welchii, Bacillus phlegmonis emphysematosae, Bacillus perfrin-

Table 43

RELATIVE FREQUENCY OF INFECTION WITH THE WELCH BACILLUS GROUP IN WAR WOUNDS

Weinberg and Sequin

Name of anaerobe	Number of deaths due to anaerobes			Total number of cases showing anaerobe
	Single species	Associated with an anerobe less pathogenic	Total	
B. welchii	0	10	19	91
B. oedematiens	3	9	12	33
Vib. septique	1	3	4	12
B. fallax		1	1	26

B. welchii+B. oedematiens....................................2

B. welchii+Vib. septique+ B. fallax............................1

gens, Bacillus enteriditis sporogenes) was discovered by Welch and Nuttall (2) in 1892. The blood vessels of a patient suffering from an aortic aneurysm showed at autopsy numerous gas bubbles. The organism is a common inhabitant of the intestinal tract of man and animals. In certain diseases it is present in excessive numbers in the digestive tract of man, causing a severe anemia. It is 1.0 to 1.5 μ wide by 4.0 to 8.0 μ long. It occurs singly or in pairs and occasionally in long threads or chains. Capsules are present on specimens taken directly from the animal body. It is non-motile. Spores when present are oval, central or excentric; very often specimens from the animal body will not show spores, however. It forms an exotoxin which is capable of rapid destruction of tissues. Four different types of the organism are listed acording to fermentation reactions.

Clostridium oedematis-maligni (Virbrion septique Pasteur; Bacillus oedematis maligni Koch) was dicovered by Pasteur (3) in 1877 associated with infections of animals which were supposed to have died of anthrax. Koch and Gaffkey later isolated the organism and studied it carefully. It is considered that Bacillus III of van Hibler and the bacillus of Ghon and Sachs are identical with it. The bacillus is 0.8 to 1.0 μ broad by 2 to 10 μ or more long, occurring principally in long chains. The spores are oval and excentric, and are very resistant. The bacillus is motile and does not form a capsule. It produces a powerful exotoxin. It is the organism usually met with in cases of malignant oedema of cattle.

Clostridium oedematiens (Bacillus neigeux, Gas oedema bacillus of Aschoff) was isolated by Weinberg and Sequin (4) in 1918 as a new species. It is closely allied to Clostridium novyi with which it is considered identical by some workers. It is 0.8 by 1.0 broad and 4 to 5 µ long, with rounded ends, occurring singly or in pairs and is somewhat motile. It is a strict anaerobe growing with great difficulty and forming abundant central or subterminal spores. It produces an active soluable toxin which is virulent for all laboratory animals.

Clostridium fallax was discovered by Weinberg and Sequin (5) in 1915 associated with other anaerobes in war wounds. It produces a toxin which is only slightly active.

Malignant oedema of animals is caused by the same group of organisms in a manner similar to that found in man. The horse is most susceptible, the affection appearing as a complication of surgical or accidental wounds. Sheep are often affected after castration or wounds inflicted while shearing. Cattle are not as susceptible to the disease but occasionally develop it following parturition. Dogs sometimes are infected through wounds inflicted while fighting. Guinea pigs, rabbits, mice, etc., are very susceptible to injection of any of these organism. The affection in animals is characterized by a hot painful, crepitating swelling at the point of inoculation. The outcome is usually fatal. Large numbers of gas bubbles are found on post mortem examination in the subcutaneous tissue surrounding the point of infection.

Gas gangrene in man is primarily a war-time affliction. Soils heavily fertilized with animal or human excreta contain numerous spores of the offending organisms with which gunshot wounds become contaminated. The depth of the wound as well as the introduction of other organisms influence the extent of the resulting oedema.

At the site of the wound an extensive hemorrhagic oedema of the subcutaneous tissue develops, the serous effusion from which is frothy with gas production and has a foul odor.

Taylor (6) has classified the various stages in the course of gangrene as follows: 1, dormant stage; 2, stage of acute gaeous distention; 3, explosive stage; 4, stage of systemic intoxication; and 5, stage of septicemia.

Christopher (7) states that there is no single pathognomonic clinical sign or symptom of gas gangrene. The condition is a syndrome and the diagnosis generally depends on the combined presence of several signs or symptoms. He gives the more reliable factors in diagnosis as history of recent wound, swelling, increased frequency of pulse rate, tympany, crepitus, pale color of skin, odor, ability to milk gas bubbles out of wound, thin discharge, roentgenographic findings, and maintained intelligence of the patient.

The laboratory may be of assistance in making bacteriological examinations of an open wound early in the disease to determine the type of anaerobe present, in order that the correct specific serum may be given, and later in the disease to determine the number of organisms present in order that closure of the wound may be performed with intelligence.

The prevention of gas gangrene depends of course upon cleanliness of all wounds. That this is accomplished under ordinary conditions in a very satisfactory way, as far as this affection is concerned, is evidenced by the almost total absence of reference to the affliction in mortality reports of civil life. At times of disaster, such as tornadoes and in war, the control of the disease is often difficult to accomplish. Its cure rests upon surgical measures often requiring amputation of the affected part. In recent years antitoxins have been developed against several of the anaerobes which are quite efficacious. The Welch bacillus serum is standardized by the United States Public Health Service and may be easily obtained. Serums against the other anaerobes are not generally available on the market.

Items of Note

1. Gas gangrene is an affection of man caused by certain anaerobic spore forming organisms.
2. The more important organisms are Clostridium welchii, Clostridium oedematiens, and Clostridium oedematis-maligni.
3. The organisms are found in the intestines of many animals, as well as of man.
4. Infection of man takes place by way of wounded tissue.
5. The affection is almost entirely limited to war wounds.
6. Malignant oedema in animals is caused in a similar way by the same group of organisms.
7. The prevention of the disease rests upon the cleanliness of wounds.
8. The cure of the disease rests upon surgery, together with the specific antitoxin.

BIBLIOGRAPHY

1. Weinberg, M., and Sequin, P.
   La gangrène gazeuse, Masson et Cie, 1918, Paris.
2. Welch, W. H., and Nuttal, G. H. F.
   Johns Hopkins Hospital Bull., 1892, *3,* 81.
3. Pasteur, Louis
   Bull. Acad. de Med., 1877, Ser. 2, 781.
4. Weinberg, M., and Sequin, P.
   Comp. rend. Soc. de Biol., 1918, *81,* 184.
5. Weinberg, M., and Sequin, P.
   Comp. rend. Soc. de Biol., 1915, *78,* 686.
6. Taylor, K.
   Jour. Path. and Bact., 1916, *20,* 384.
7. Christopher, F.
   Jour. Am. Med. Asso., 1919, *72,* 407.

SECTION FIVE

A REVIEW OF THE ROLE PLAYED BY EACH ANIMAL IN THE SPREAD OF DISEASE

Section Five

A REVIEW OF THE ROLE PLAYED BY EACH ANIMAL IN THE SPREAD OF DISEASE

Chapter XXVII

THE ROLE OF CATTLE, HORSES, SWINE, SHEEP AND GOATS, DOGS, CATS, RATS AND MICE, POULTRY AND BIRDS AND WILD GAME

"Man is his own worst enemy" in the spread of communicable diseases but the lower animals are a close second. In the early stages of his existence man came in contact with animals chiefly in the hunt; his chances of infection from them were consequently rather remote except as he happened to encounter a sick animal. As soon as man ceased his nomadic wanderings and established permanent habitations his troubles began. Not only did the animals which he domesticated transmit to him deadly disease, but rodents took up their abode with him also, with all consequent ill results. As far back as history is recorded in writing, mention is made of some of the diseases which we have since learned to connect with the lower animals.

Cattle. Cattle have been the most indispensible animals of all the domesticated creatures, furnishing not only labor but meat and milk. The cow has been well defined as the "foster-mother of the world." Because of man's intimate contact through milk and meat supply, she has been responsible for considerable human woe, so much that the beast has been called a "pest to be found wherever civilized man makes his abode" (1).

The ancestry of our domestic cattle goes back to prehistoric times. Wild cattle of one species or another were common in many parts of the world and were hunted by wild tribes for food. Very early, however, man apparently domesticated the animal which since has been developed into our milk cow and our beef steer.

In the United States in 1927 there were 57,521,000 head of cattle on farms, of which number 21,824,000 were milk cows.

There were slaughtered in 1926 at regularly established slaughter houses under Federal inspection 10,180,000 cattle and 5,153,000 calves. In addition to this, numerous cattle and calves were slaughtered on the farms and at slaughter houses not under Federal inspection.

Twenty communicable diseases of cattle are listed by Mohler, (2) thirteen of which have no concern for man. The latter are contagious pleuropneumonia, rinderpest, hemorrhagic septicemia, blackleg, necrotic stomatitis, malignant catarrh, malignant oedema, Texes fever, chronic bacterial dysentery, nagana, cattle farcy, white scours of calves and infectious ophthalmia. There are likewise a score of animal parasites of cattle, almost all of which are of no importance to man. There are four parasites of the stomach, two tapeworms of the intestine with one large round worm and several small round worms, two flukes of the liver and lung, three kinds of tapeworm cysts of the liver and lung, several other lung worms, flukes of the large vein and a roundworm of the eye, none of which find shelter in man.

The bacterial diseases of cattle which are communicable to man are tuberculosis, anthrax, contagious abortion, foot-and-mouth disease, rabies, cowpox and actinomycosis. The only animal parasite which man contracts from cattle is the beef tapeworm, *Taenia saginata*. Human diseases with which cows may become afflicted and pass back again to man are scarlet fever, septic sore throat and diphtheria. Smallpox may be contracted from human beings by cattle in the form of cowpox, but modified to such an extent that it never again produces smallpox.

In relative importance to man bovine tuberculosis outranks all other affections, causing more trouble than the rest of the strictly animal diseases combined. Septic sore throat is second in importance, being responsible for large epidemics from time to time. Contagious abortion, which apparently infects man more often than has been supposed, ranks third, and anthrax fourth. Foot-and-mouth disease, rabies, diphtheria and scarlet fever are infrequently passed to man, while it is doubtful whether actinomycosis is passed directly to man from cows or not. Cattle suffer from Salmonella infections which cause food poisoning in man and they sometimes harbor in their intestines the organisms of botulism, tetanus and gas gangrene.

Horses. The horse is counted among the most intelligent of the lower animals. His friendship for man has been close and his contact intimate, yet the instances when he has conveyed a communicable disease to his master have been comparatively rare.

In early times the wild ancestors of the modern horse roamed in bands over the territory between Northern Africa and Eastern Manchuria. They were probably the first animal hunted by man for food and the first one tamed. The oldest carvings left by the inhabitants of the valley of the Euphrates showed that saddle horses were familiar to them. It is said that the use of the horse by the people of central Asia gave them a supremacy over the unmounted tribes. Much later, about 2,000 B.C., the horse came into general use in Arabia and Egypt, where the camel and ass had been used before.

The number of horses in the United States has been diminishing during the last few years with the increased use of motor transportation. In 1918 there were 21,555,000 horses in the country while in 1927 there were only 15,279,000. The control of contagion in horses has been made easier not only by the lessened number of such animals, but also by the fact that they have almost disappeared from city streets. The horses that remain are largely on the farms where they lead lives more or less isolated from other animals of their kind and hence have less opportunity than in previous times of contracting contagion.

Fourteen communicable diseases of horses are listed by Huidekoper (3), most of which have no significance for man. Influenza, contagious pneumonia, strangles, purpura hemorrhagica, dourine, infectious anemia, surra and osteoporosis have never been known to infect man. Horsepox is related to cowpox and may be contracted by man, but passage through the human so modifies it that the horse is difficult to infect again with the same virus. Infectious abortion is caused by *Salmonella abortus-equi,* an organism of the food poisoning group, (not to be confused with *Brucella abortus* causing contagious abortion in cattle). While a few epidemics of food poisoning in man have followed the consumption of horse meat in Europe the connection between this organism and the disease in man lacks confirmation. Sporotrichosis of horses is not uncommon in the United States but according to Meyer (4) there is no reason to believe that man is infected from this source. The absence of the disease among farmers and veterinarians in Pennsylvania, where the infection is prevalent among horses, lends weight to his argument.

Glanders is the only disease to which man is subject that finds its reservoir in the horse. Veterinarians and laboratory workers are the two groups of persons most likely to become infected. Anthrax infects horses in those communities where the disease is prevalent among other animals. The hide or the hair of such infected animals is dangerous to man as in the case of shaving brushes made from improperly treated horse hair. Rabies is infectious for horses but rarely is the disease passed on to man. The virus of foot-and-mouth disease is closely related to that of vesicular stomatitis of horses, but horses are entirely resistant to the former.

Tuberculosis is rather a rare disease of horses as is also actinomycosis. Infection with *Brucella melitensis* of goat origin or *Brucella abortus* of bovine origin, while it has been found to exist, is probably so rare as to be disregarded. Horses are resistant to the rodent diseases, although under exceptional conditions experimental infection with plague and Rocky Mountain spotted fever has taken place. The horse may harbor ticks of the Dermacentor variety which transmit both Rocky Mountain spotted fever and tularemia. Persons pulling ticks from horses are thus exposed to infection. No intestinal parasites are transmissable from horses to man.

Swine. Swine are of ancient descent. They have been developed from the wild boar of Asia, being domesticated in China about 2900 B.C., and in Europe about 1500 B.C.

Swine are responsible for a number of human infections. While they do not have the intimate contact of the home in this country that they do in some places, indirectly they form contact sufficiently to transfer several diseases. In the United States in 1927 there were 52,536,000 swine on the farms. In 1926 there were 40,636,208 that were slaughtered at establishments under federal supervision.

The round worm, *Trichinella spiralis,* together with the tapeworm, *Taenia solium,* have influenced the course of man's actions for thirty centuries. They were responsible for the ancient Jewish prohibition against the use of pork and as recently as 1880 and 1890 they caused international complications between the United States and Germany. The latter country refused to import pork from this country because of reputed infestation with trichina. Hogs are as susceptible to human tubercle bacilli as they are to bovine. Care must be taken therefore in feeding to hogs garbage which may have been contaminated by a tuberculous person. Contagious abortion of hogs is caused by an organism slightly different from *Brucella abortus* of cattle and which has for man more virulence than does the bovine organism. McAlpine draws attention to the fact that undulant fever in man is most prevalent in the United States in those districts where the hog raising industry is greatest. Hogs may also contract on occasion anthrax, foot-and-mouth disease, milk-sickness and actinomycosis. Glanders infection is rare. Organisms of the food-poisoning group, especially *Salmonella suipestifer,* very often affiict hogs. Swine erysipelas may be transmitted from hog to man.

Sheep and Goats. Sheep, in their relation to public health, are of minor significance. While they are very susceptible to anthrax infection, other animals, both domestic and wild, share with them the responsibility of perpetuating the disease. Infected sheep skins and wool are industrial hazards and as such have received particular attention. Sheep are susceptible to foot-and-mouth disease but that disease is comparatively rare in this country. Rabies is contracted from rabid dogs but is seldom passed on to other animals or man. The liver fluke, *Fasciola hepatica,* and the dog tapeworm, *Taenia echinococcus* may pass part of their life histories in sheep. Tuberculosis is seldom found in sheep, either of human, bovine or avian origin. Food poisoning epidemics have been traced to sheep suffering from infection with *Salmonella aertrycke.*

Goats are in very much the same category with sheep in regard to the public health, with the exception of undulant fever. Without showing any signs of infection, they may excrete thousands of virulent organisms of *Brucella melitensis* in the milk. In the warm countries around the Mediterranean Sea this has been a serious problem; in the United States goats have not been used for milk purposes in sufficient numbers to be of practical importance. An extensive epidemic of undulant fever in Arizona in 1922, however, emphasized the need of pasteurizing goat's milk. Goats are somewhat more susceptible to bovine tuberculosis than are sheep. Rabies, anthrax and foot-and-mouth disease may be contracted but are seldom passed to man.

Dogs. The dog of all animals enjoys the most intimate contact with man, sharing not only his dwelling, but sometimes even his bed and his board. Man is exposed therefore to several of the ailments to which dogs are heir.

The origin of the modern domestic dog is impossible to follow, but it is probably of composite descent. Wild dogs of one species or another have been found universally and have been tamed by practically all primitive tribes.

The number of dogs in the United States is impossible to estimate definitely, for many owners have failed to register their animals. There are numerous dogs, however, which can boast no master but wander from the hospitality of one kind hearted person to another or else live on the scraps of the street and garbage pail, a menace not only to all other dogs but to mankind as well. Buckley considered in 1926 that the number of dogs in the country was greater than at any previous period (5).

Rabies is the most serious of the afflictions which can be charged against the dog. In no other animal does the disease find a reservoir for its perpetuation (save possibly the skunk) and seldom do other animals spread it farther than one remove from the dog. The increased number of dogs together with rapid modes of transportation of both man and his dog has resulted in a sharp increase in rabies not only in the United States but many other countries.

Animal parasites which infest most dogs form somewhat of a menace to man. The most dangerous is *Taenia echinococcus* which carries on its adult stage of life in the intestine of dogs while its larva stage is spent in the tissues of man or other animals. Another tapeworm which should be mentioned is *Dipylidium caninum.* Its larva stage occurs in lice and fleas which infest dogs, children becoming infected by accidently swallowing such lice and fleas or crushing them and afterwards sucking the fingers. The guinea worm, *Dracunculus medinensis,* has been found in both dog and man while several liver flukes of dogs may be transferred to persons. Three forms of mange afflict dogs, only one of which is transmissible to man—sarcoptic. Among the four forms of ringworm from which dogs suffer, a rare case is passed to persons.

Dogs may become infected with various bacterial diseases from other animals but do not form a serious menace to man. Tuberculosis is contracted sometimes from association with tuberculous persons, from drinking infected cows milk or from the offal of slaughter houses. Anthrax and glanders are sometimes contracted by dogs from eating animals that have died of those diseases. Infection with foot-and-mouth disease is difficult as it is also with trichinosis. Dogs are not generally susceptible to the rodent diseases, but they may act as mechanical carriers of rat-bite fever, where infection has followed the bite of a dog which had recently fed on an animal dead of the disease. The same is possible with other rodent diseases (6).

Dogs have been found susceptible to the organism causing scarlet fever and free access of a dog to a human case of the disease might be a menace to other children.

Cats. Cats enjoy an intimate contact with the human family. Because of prolific breeding and the mistaken sympathy of some persons which allows cats to wander homeless instead of being destroyed, the number of such animals in the country is numerous. Cats are fairly resistant to many of the human diseases. On rare occasions they have been found tuberculous from infected milk or meat. They are only slightly susceptible to foot-and-mouth disease and the Berlin commission found them entirely refractory. Cats gave positive agglutination tests for undulant fever in the Island of Malta and Bruce isolated *Brucella melitensis* from the mesenteric glands of five such infected animals. They are susceptible to glanders, but rarely contract the disease naturally. They may become infected with rabies from contact with rabid dogs, forming a serious menace to children. Cats are hosts of several animal parasites, some of which are transferable to man. The liver fluke, *Opisthorchis felineus,* is common in Europe while the closely related species, *Opisthorchis pseudofelineus* is found in central parts of the United States. Both parasites are natural inhabitants of the cat from which man is infected. The intestinal flagellate *Giardia* found in rats and mice is a variety of the human species, and cats have been found responsible in disseminating the infection. The dog tapeworm, *Dipylidium caninum,* is harbored by cats also, as is the hog round worm *Trichinella spiralis.* Cats are relatively immune to the rodent diseases. They show slight susceptibility to plague and to tularemia but are relatively unimportant in the spread of each disease. Several instances are recorded where rat bite fever followed the bite of a cat, but probably the cat was a mechanical carrier of the spirochetes picked up from eating an infected rat. While the relation of cats to diphtheria is a disputed point, Simmons reports two cats from which he obtained diphtheria bacilli.

Rats and Mice. The rat, of all useless animals, is the most detestable. As a marauder he is bad enough, as a murderer he excels. Originating in Asia as did man himself, he has followed man in his travels to the uttermost parts of the earth. The number of rats in any city is impossible to estimate, but it is safe to say that there at least as many rats as there are people; on the farms there are countless thousands more.

The brown rat or Norway rat is the most common species in the United States. It is larger and more ferocious than either the black rat or the roof rat. It appeared in this country about the time of the American Revolution, probably being brought from England as an accidental passenger (it is not a natural inhabitant of ships as are the other two species). It has made itself at home here, in houses and barns in the country and in warehouses and about markets and wharves in the city. It lives in the ground, in excavations or under floors, driving the other rats to the upper floors of buildings. It often uses the sewers in cities for highways, hence the name "sewer rat." It breeds prolifically, the female, after a gestation period of twenty-one days, giving birth to a dozen or more young. From three to five litters a year occur, usually during the warm months of the year. Some females, however, may produce more litters, and it is not uncommon to find young rats in cold weather.

The black rat or English rat has been known in Europe for centuries. It was common in this country with the early settlers, but it has since been driven out of most communities by the brown rat. It is a great sea-traveler, breeding in the walls or in the cargo of vessels.

The roof rat or Egyptian rat is thought to have originated in Egypt. It is similar in habits to the black rat, including its seagoing propensities. It is not common in this country, however, because of the animosity of the brown rat. In warm climates it nests in trees, as does also the black rat, and hence holds its own against the brown rat.

The common house mouse is closely related to the rats, the principle distinction being that of size. It is found everywhere, being able to hold its own against the brown rat because it can escape through holes too small for the rat to enter. It does not seem to play as important a role in the dissemination of disease as does the rat.

The most serious indictment against the rat is the spread of bubonic plague. In the fifth century B.C. the Philistines lost from plague 50,000 of their number; in the fifth century A.D. the mortality from plague rose to 10,000 persons daily; in the fourteenth century 25,000,000 people, or a fourth of the world's population perished from this disease alone; in the present pandemic more than 10,000,000 in India have died from plague, not to count the deaths in other countries. The total number of deaths due to plague since the beginning of history would run into hundreds of millions and possibly billions (8).

Trichinosis is charged against the hog as far as human infections are concerned, but the rat is responsible for perpetuating the disease and infecting swine. But for the rat, the disease could be eliminated.

Rat-bite fever in man is usually the result of a rat bite, while spirochetal jaundice is conveyed from the rat either by the bite or other close associations. Tularemia has been found in rats and mice on several occasions.

Numerous food poisoning epidemics are chargable to rats. *Salmonella enteriditis* is often an inhabitant of the rat intestine. Food to which rats and mice have access may become contaminated with the droppings. Rat viruses for the destruction of rats have been responsible for human infections also. The Hooper Foundation for Medical Research (9) gathered rats from various places in San Francisco. Among 775 animals examined, 58 of them were infected with bacterial diseases which could be transmitted. Two per cent carried in their intestines bacteria of the food poisoning group, and were capable of infecting food by their droppings.

Among the animal parasites other than trichina, there are several which rats and mice may harbor. The intestinal protozoa of man may be picked up and carried from contaminated food or water. This is especially true of *Entamoeba histolytica*. *Giardia* of rats and mice is probably a variety of the human species and hence of danger to man. Rats may eat the eggs of the dwarf tapeworms *Hymenolepsis nana and Hymenolepsis diminuta,* and thus become infected. The same is true of *Ascaris lumbricoides.*

Poultry and Birds. Of livestock on the farms, poultry is the most numerous. In 1925 the receipts from poultry and eggs in the United States was estimated at $1,000,000,000—about the same as for cattle. The diseases to which poultry is heir are numerous, but are of little importance to man. Avian tuberculosis is the most serious; but the number of human infections are small compared to those from the bovine or human strains of the tubercle bacillus. White diarrhea is difficult to control in poultry, since the organism, *Salmonella pullorum,* is transferred through the egg. The menace of such infected eggs has been demonstrated experimentally by feeding laboratory animals (10), but no records of human infections have been collected. Diphtheria may be contracted by poultry from a human case and thus form a menace to susceptible persons who may come in contact with such birds. Birds other than poultry are of little consequence in the spread of contagion. Parrots are susceptible not only to all forms of tuberculosis, but also to psittacosis, but the number of parrots in the country is so small as to make them of relatively small importance. The wild birds sometimes act as mechanical carries of infection, such as anthrax, from feeding on animals dead of the disease.

Wild Game. Wild game is responsible for spreading several diseases communicable to man. Plague in America is perpetuated mainly by the California ground squirrel while in Asia the tarbagan and in India the musk shrew play similar rôles. In England the rabbit and hare have been found naturally infected with plague (11). Tularemia finds its reservoir in the rabbit and hare and is transmitted to man either directly by contact or through insects. Rocky Mountain spotted fever is carried by the tick *Dermacentor,* the larval forms of which are harbored by the ground squirrel, wood chuck and similar small game,. while the adults are carried by the Rocky Mountain goat, bear, lynx, badger, coyote, etc. Many wild animals harbor trematodes, cestodes and nematodes which may affect man upon occasion. Scott reports an epidemic among baboons from which *Salmonella aertrycke* was isolated in 12 out of 16 fatal cases. Wilbert (12) studied an epidemic among chimpanzees which simulated Weil's disease in which he found spirochetes similar to *Spirocheta icterohemorrhagica.* Foot-and-mouth disease in the 1924 epidemic in California spread to the deer in the forests, making it necessary to hunt down and kill more than twenty thousands such animals. Anthrax was said to be propagated and spread by deer in the early settlement of Louisiana by the French.

BIBLIOGRAPHY

1. Dawson, F. W. W.
Jour. State Med., 1923, *31,* 282-288.

2. Diseases of Cattle
Revised Edition 1923, p-358, U. S. Depart. of Agriculture.

3. DISEASES OF THE HORSE
Revised Edition 1923, p-507, U. S. Dept. Agriculture.

4. MEYER, K. F.
Jour. Am. Med. Asso., 1915, *65*, 579-585.

5. YEARBOOK OF AGRICULTURE, 1926, p-624.
U. S. Dept. of Agriculture.

6. For further discussion see
Cameron, T. W., Lancet, 1922, *1*, 564-565. Joannides, M., and Riley, W. A., Jour. Am. Med. Asso. 1925, *85*, 1054-55.

7. THE RAT AND ITS RELATION TO PUBLIC HEALTH
Public Health Bull. No. 30. 1910, U. S. Public Health Service.

8. LLOYD, B. J.
Jour. Am. Med. Asso., 1925, *85*, 729-733.

9. ANON
Jour. Am. Med. Asso., 1927, *88*, 1976.

10. RETTGER, L. F., HULL T. G., and STURGES, W.S.
Jour. Exp. Med., 1916, *23*, 475-489.
Also RETTGER, L. F., SLANTZ, C. A., and McALPINE, J. G.
Storrs Experiment Station Bull. 140, 1926.

11. MANSON—BAHR, P. H.
Vet. Rec., 1926, *6*, 603-611.

12. ANON.
Jour. Am. Med. Assoc., 1927, *89*, 1529.

INDEX; AUTHORS

INDEX OF AUTHORS

A

Abbott, A. C .
  diphtheria in cats, 260
  diphtheria in cattle, 261
Adamski
  spirochetal jaundice, 226
Aiello
  resistance of B. anthracis, 41
Alfort school of veterinarians
  glanders
    spontaneous origin of life and non-infectiousness of, 110
American Commission
  foot-and-mouth disease
    cultivation of organism, 57
    similarity to organism causing vesicular stomatitis in horses, 57
    resistance of in man, 60
Anderson
  tetanus, 282
Andervont, H. B.
  epithelioma contagiosum virus, 104
Andrews, J. B.
  anthrax—deaths from in U. S., by occupation from 1910-1917, table of, 46
  anthrax—human, records of fatal cases in U. S. from 1910-1917, 44
  anthrax by occupation, table of, 45
  anthrax as to occupation, 47
Anthony
  glanders, agglutination test, 116
Apsyrtus
  glanders, 110
Aretaeus
  tuberculosis, 1
Aristotle
  glanders, 110
  rabies, 118
Arizona
  foot-and-mouth disease, "border war" against, 54
Armstrong, C.
  diphtheria in cattle, 262
  post-vaccination tetanus, 108
  scarlet fever, 266
  septic sore throat, 255
Arnold, L.
  food poisoning, predisposing factors, 165, 166
Ashby, A.
  diphtheria in cattle, 261
Aurelianus, Celius
  rabies in man, 118
Avery, O. T.
  septic sore throat, 255

B

Babes
  glanders, 111
  rabies, 139
Bachman, G. W.
  test for trichinosis, 179
Bachman, F. M.
  hemolytic strepticocci in milk, 253
Bachem
  psittacosis, 152
Baillie
  tuberculosis, 2
Bainbridge
  food poisoning due to B. enteriditis and B. suipestifor, 157
Bang
  avian tuberculosis, 23
  Bacillus abortus, 65, 79
  contagious abortion, 65
  discovered Bacillus abortus, 64
  streptococcus infection, 250, 251
  swine erysipelas, 170
Banker, A. J.
  rat-bite fever, 231
Bannerman
  plague, 201
Bardach
  rabies virus in milk and meat, 149
Barlow
  contagious abortions, 64
Barthelemy
  anthrax, 40
Bauer, J. H.
  tetanus, 283
  tetanus in man, 280
Bauchard
  glanders, 111
Bauermeister
  swine erysipelas, 172
Bayle
  tuberculosis, 2
Beals, L. H.
  vaccination against plague, 206
Beatty, N.
  deaths from rabies, 138
  statistics on world prevalence of rabies, 124
Belyea, G. N.
  abortus bacillus, 76

Bengston, I. A.
  tetanus organisms on scarifiers, 107
Bergey, D. H.
  acid-fast bacteria, 4
  actinomycosis, 94
  Salmonella group, 158
  Streptococcus pyogenes, 251
Bergey's Manual of Determinative Bacteriology
  alcaligines, 66
Berlin Commission
  foot-and-mouth disease, 56
  foot-and-mouth disease in cats, 299
Bettison, Wm. L.
  trichinosis in man, 178
Bigelow, A. H.
  septic sore throat, 255
Billing
  smallpox, 103
Black, L. A.
  undulant fever, human infection, 80
Blake, F. G.
  rat-bite fever, 231
Bloomer, G. T.
  typhoid fever in chickens, 248
Blumer, G.
  infectious jaundice, 222
Boerner, F.
  cowpox, 103
Boez, L.
  foot-and-mouth disease in guinea pigs, 56
Bollinger
  actinomycosis, 94
  glanders in man, 113
Bongert
  tuberculosis, 20
Boreggi
  anti-rabic vaccination, 130
Bosquillon
  rabies infection, 120
Boston Board of Health
  streptococcus infection, 250
Boström
  actinomycosis, 94
Bovaird, D.
  mortality in trichinosis, 178
  trichinosis in rats and man, 177
Bowley
  glanders, 111
Brandt
  diphtheria in dogs, 263
Brauell
  anthrax bacillus, 40
Breschet
  identified human rabies with diseases in dog, 119
Bricker, E.
  anthrax, 47
British Commission
  foot-and-mouth disease, cultivation of organism, 57
British Commission for the investigation of Mediterranean Fever
  Malta fever, 63, 72
  undulant fever, 77
British Ministry of Health
  anthrax—report on, 50
British Royal Commission
  on Tuberculosis, 23
  tuberculosis, 13
Bristow, A. T.
  glanders, vaccination against, 114
Brodie, T. G.
  diphtheria in cats, 260
Brooks, R. S.
  plague, optimum temperature, 198
Brophy, T. J.
  food poisoning, predisposing factors, 166
Brown, J. T.
  bovine mastitis, 254
  hemolytic streptococci, alpha and beta types, 251
  septic sore throat, 255
Brown, T. R.
  milk sickness, 87
Brown
  smallpox, 103
Brown, Elsie W.
  tuberculosis, 13
Brown, J. H.
  Streptococcus epidemicus in milk, 252
Bruce
  Malta fever
    isolated Micrococcus melitensis, 63
  micrococcus melitensis, 65
  undulant fever in cats, 299
Buchanan
  glanders, 116
Buchanan, G.
  spirochetal jaundice, 227, 223
Buck, J. M.
  contagious abortion in bulls, 73
Buckley
  dogs, 298

Bullock
  tuberculosis, 13
Bundesen, H.
  method of detecting streptococcus epidemicus in milk, 252
Burke
  botulinus organism on cherries, 276
Burnet
  diphtheria in monkeys, 263
Burnett
  Malta fever
    "remittant malarial fever," 63
Busey
  scarlet fever in cows, 267
Busson
  resistance of B. anthracis, 41
Butler, W. J.
  human tuberculosis in hogs, 26, 271
Buzello
  tetanus in man, 280
Brüll
  contagious abortion, test for, 64
Bräuer
  contagious abortion, 64

C

Caldwell, F. C. and E. L.
  ascaris infection, 181
Calkins, G. N.
  rabies, protozoan theory, 128
Calmette, A.
  avian tuberculosis, 22, 23
  tuberculosis, 32
  tuberculosis, vaccination of calves, 35
  tuberculosis, immunization against, 35
  tubercle bacilli, 3
  tuberculosis in horse, 7
Cameron, J.
  scarlet fever in cows, 266
Capps, J. A.
  mastitis, 253
  septic sort throat, 257
Capitan
  glanders, 111
Carr
  bovine mastitis, 254
Carpenter, W. M.
  anthrax, 41
Carpenter, C. M.
  abortus agglutination tests, 80
  Brucella, 66
  mammitis, 253
  undulant fever from raw milk, 82
  undulant fever, human injection, 80
Caspard de Saunier
  glanders, 110
Castellani
  alcaligines abortus, 65
  alcaligines melitensis, 65
  anthrax spores, 43
  plague, 201
  world distribution of undulant fever in man of goat origin, 68
Cartwright
  swine erysipelas, 170
Catlin
  smallpox, 101
Cowan, G. W.
  rocky mountain spotted fever, 241
Celsus
  rabies in man, used term hydrophobia, 118
Cesari, E.
  agglutination tests, abortus, 70
Chabert, P.
  anthrax, 39
Chalmers
  Alcaligines abortus, 65
  Alcaligines melitensis, 65
Chapin, C. W.
  Bacterium tularense, 209
  vaccination against plague, 206
Charin
  glanders, 111
Chauveau
  tuberculosis, 20
Chicago Health Dept.
  method of detecting streptococcus hemolyticus in milk, 252
Chowning, W. M.
  rocky mountain spotted fever, 236, 238, 240
Christiansen, M.
  swine erysipelas in lamb, 171
Christopher, F.
  gas gangrene, 287
Cobbett, L.
  diphtheria in horses, 263
  tuberculosis, 6, 13
Coffee
  malta fever, 73
Coleman
  glanders, 111
  glanders from horse to donkey, 110

Coles, A. C.
  spirochetal jaundice, 223
Committee on Bovine
  infectious abortion, 73
Committee on the Control of Communicable Diseases, 247, 248
Connecticut State Dept. of Public Health
  scarlet fever streptococci of human origin, 267
Cook
  rat-bite fever, 231
Cooledge, L. H.
  abortus bacilli in milk, 79
Cooper
  cause of food poisoning, 163
Copeman, S. M.
  smallpox in monkeys, 104
  smallpox transmission, 103
Costill
  rabies, cauterizing of wounds, 143
Cotten, W. E.
  contagious abortion, 64
  undulant fever, 79
Crohn, B. B.
  incubation period in rat-bite fever, 233
  rat-bite fever, 231
Cumming
  anti-rabic vaccination, 133
Couch, J. F.
  milk sickness, isolated trematol, 87, 89
  milk sickness, 92
Cragg, F. W.
  plague, 203
Craig, C. F.
  malta fever, symtoms of, 76
  undulant fever caprine origin, 78
Cramp, W. C.
  glanders, vaccination against, 115
Creech, G. T.
  contagious abortion in bulls, 73
  swine erysipelas, 170

D

Dade
  tularemia, 214
Dahmen
  foot-and-mouth disease, Loeffleria nevermanii organism, 57
Damon, S. R.
  cause of food poisoning, 164
Danforth, T. F.
  tubercle bacilli in milk, 17
Davis, D. J.
  hemolytic streptococci, beta type, 251
  mastitis, 253
  streptococcus epidemicus, 251, 252
Davaine, C.
  anthrax bacillus, 40
Dean, A.
  diphtheria in cattle, 261
Delafond
  anthrax, 40
DeNobele
  isolated salmonella aertrycke, 161
Dick, G. F.
  rat-bite fever, 233
  streptococcus scarlatinae, 266
Dickson, E. C.
  botulism in man, 277
  botulinus organism on cherries, 276
Dieter, L. V.
  tularemia, 214, 216
Dochez
  streptococcus scarlatinæ, 266
Doi
  vaccination of dogs against rabies, 145
Douglas
  rat-bite fever, 232
Douville, M.
  tuberculosis in dog, 8
Drago
  resistance of B. anthracis, 41
Dubois
  agglutination tests, abortus, 78
  malta fever, extent of abortion, 69
Dujardin
  trichinosis, 175
Dunedin
  tularemia, 214
Dupuy
  mortality of psittacosis, 153

E

Earle, C. W.
  rat-bite fever, 231
Easton, E. J.
  botulism, 276
Eastwood
  tuberculosis, 13
  tuberculosis in hogs, 26

Edwards
  malta fever, 73
Eggstein, A. A.
  spirochetal jaundice, 223
Eichhorn, A.
  anthrax from tannery wastes, 42
  Bacillus abortus , micrococcus malitensis, 65
  deaths from rabies, 138, 139
  glanders, diagnosis of, 115
  malta fever, 72
  spore vaccine against anthrox, 48
  vaccination of dogs against rabies, 145
  vaccinated dogs in U. S., 147
Elder, C.
  avian tuberculosis, 23
Elfer
  avian tuberculosis, 24
English Royal Commission
  tuberculosis in hogs, 25
von Ermengan, E.
  discovered botulinus baccillus, 275
Evans, Alice
  Brucella, 66
  Bacillus abortus in cows milk, 65
  contagious abortion identity of abortus and micrococcus melitensis, 64
  on identiy of abortus and melitensis organisms, 80
  melitensis-abortus group serological classification, 66
  melitensis, 70
  malta fever, 67
  undulant fever, susceptibility of man, 82
  undulant fever, porcine origin, 82
  undulant fever and pasteurization of milk, 83
  undulant fever, human infection, 80
Evans, W. A.
  rat-bite fever, 231
Ewing
  actinomycosis, 97

F

Fabricius
  rabies, 140
Federal Government
  control of vaccines, 54
  eradication of tuberculosis in animals, 28
  post-mortem inspection of meat, 25
Ferran
  anti-rabic vaccination, 130
Ferenbaugh, T. L.
  malta fever, 72
  undulant fever-endemic, 78
Fife, C. A.
  ascaris infection in man, 181
Finkler
  psittacosis, 152
Fischer
  vaccinia from smallpox, 103
Fitch, C. P.
  avian tuberculosis, 22
Ford
  smallpox, 103
Fornet
  smallpox, 102
Forsbeck, F. C.
  septic sore throat, 255
Foulerton, A. G. R.
  spirochetal jaundice, 223
Fournier
  Bacillus psittacosis, 152
  "Charbon Malin," 39
Franck
  contagious abortion, 64
Francis, E.
  tularemia, 211, 212, 217, 218
  tularemia, sources of infection, 218
Fraser
  tuberculosis, 13
Freyer
  smallpox in rabbits, 104
  vaccinia from smallpox, 103
Friesleben, M.
  animals responsible for food poisoning, 164
Fricks
  prevention of rocky mountain spotted fever, 242
  rocky mountain spotted fever, 236
Frosch
  foot-and-mouth disease, filterable viruses, 57
  foot-and-mouth disease, Loeffleria nevermanii organism, 57
Frost, W. D.
  bovine mastitis, 254
  hemolytic streptococci in milk, 253

Frost, W. H.
  Streptococcus epidemicus in milk, 252
  poliomyelitis, 248
  septic sore throat, 255
Futaki, K.
  spirocheta morsus muris, 232

G

Gaertner
  enteriditis infection, 159, 160
  isolated enteriditis organism, 160
Gaiger, J. C.
  glanders in man, 113
Gaffkey
  clostridium oedematis-maligni, 286
Gage, E. A.
  undulant fever, human infection, 80
Galen
  pesta magna, 100
Geiger, J. C.
  botulism in string beans, 276
  botulinus organism on apricots, cherries and olives, 276
  food poisoning, 157
Gentry, E. R.
  malta fever, 72
  undulant fever-endemic, 78
Georgia State Board of Health
  decrease in rabies, 146
Gerlach
  swine erysipeles in man, 173
Gettinger, William
  rocky mountain spotted fever, 241
Gilbert
  bacillus psittacosis, 152
Gilliam, D. I.
  rat-bite fever, 231
Gins, H. A.
  foot-and-mouth disease wound infection, 59
Good, H. H.
  contagious abortion in swine, 75
Goodman
  diphtheria in rabbits, 263
Golaz, B. S.
  diphtheria in cattle, 262
Gordon
  identity of smallpox and cowpox, 103
Gordon, J. K.
  tuberculosis, 13
Graham, Robert
  avian tuberculosis, 23, 24
  botulism in cattle, 276
  malta fever, 73
  tuberculosis in swine, 25
Graham, M.
  diphtheria in cattle, 262
Gray, Cora E.
  tuberculosis mortality, 31
Green
  tularemia, 214
Gregory, D. A.
  undulant fever, human infection, 80
Griffith, A. S.
  tuberculosis, 13
  tuberculosis in hogs, 26
Griffith, F.
  tuberculosis, 13
Grinstead
  contagious abortion test for, 64
Grund
  glanders, agglutination test, 116
Gruner
  rabies in dogs, 119
Gulland, G. L.
  psittacosis, 153
  spirochetal jaundice, 227, 223
Guarniéri, G.
  Cytoryctes variola-vaccinæ, 101, 102
Guerin
  tuberculosis, immunization against, 35
Gumm, M.
  streptococcus in milk, 252
Gürlt
  tuberculosis, 2

H

Haffkine
  plague, 201
  vaccine against plague, 206
Halpin
  avian tuberculosis, 24
Hardenbergh, J. A.
  mastitis, 253
Hart
  malta fever, 72
Harvey
  anti-rabic vaccination, 131
  rabies, 141

Harris
  anti-rabic vaccination, 132
Harris, N. M.
  milk sickness, Bacillus lactimorbi, 87
Harz
  actinomyces, 94
Hastings
  avian tuberculosis, 24
Hata, S.
  vaccination of dogs, against rabies, 145
Heath, L. M.
  avian tuberculosis, 23
Hektoen
  scarlet fever in monkeys, 269
Helman
  mallein discovered by, 111
Henke
  diphtheria in cats, 260
Henry, J. E.
  diphtheria in cattle, 262
Herbst
  trichinosis, 175
Herndon
  milk sickness, 86
Hertwig
  rabies infection, 119
Heusinger, C. F.
  anthrax, 40
Hill, B.
  scarlet fever in cows, 266
Hilton
  trichinosis, 175
Hippocrates
  food poisoning, 156
  glanders, 110
  tuberculosis, 1
Hiscock, I. V.
  spirochetal jaundice, 226
Hoare, E. W.
  tuberculosis, 2
  tuberculosis in horse, 8
Hobmaier
  foot-and-mouth disease, 56
Hoki
  spirochetal jaundice, 223
Holt, R. L.
  malta fever, 72
Holth
  contagious abortion test for, 64
Hooper Foundation for Medical Research
  transmissible diseases in rats, 301
Horace
  food poisoning, 156
Horder
  rat-bite fever, 231
Horrocks, H. W.
  malta fever, 78
Hoy, W. A.
  tubercle bacilli in cow manure, 17
Huddleson, I. F.
  Brucella, 66
  susceptibility of animals to Br. melitensis and Br. abortus, table of, 69
  undulant fever, human infection, 80
Huidekoper
  communicable diseases in horses, 295
Hull, T. G.
  anti-rabic vaccination in Illinois in 1928, 147
  deaths from rabies, 138, 139
  failures of anti-rabic vaccination, 146
  failure of anti-rabic treatment, 146
  poliomyelitis in swine, 247
  rabies in dogs in Illinois, 148
  rabies in stray dogs, 148
  undulant fever, human infection, 80
  white diarrhea in man, 162
Huzard
  tuberculosis, 2
Huytera
  glanders, tests for, 116
Hygienic Laboratary
  rabies, seasonal prevalence of, 121
  rocky mountain spotted fever, 238
  vaccination against rabies, 130
Hübener, H.
  Spriocheta nodosa, 222
  spirochetal jaundice, 225
Högyes
  anti-rabic vaccination, 130, 131

I

Ido, Y.
  Spriocheta icterohemorrhagiae, 222
  spirochetal jaundice, 223, 224
Illinois State Legislature
  a State law against the tuberculin test, 27

Illinois State Dept. of Public Health
  rabies in animals, 35
Inado
  Spirocheta icterohemorrhagiae, 222
Inada, R.
  spirochetal jaundice, 225
Indian Plague Commission
  plague, 197, 200, 202
  vaccination, 206
Israel
  actinomycosis, 94
Ito, H.
  spirochetal jaundice, 223

J

Jansco
  avian tuberculosis, 24
Jarosch
  swine erysipelas in poultry, 172
Jenner, E.
  history of vaccination, 101
  horse pox, 104
  smallpox, 102
  smallpox in cattle, 271
Jerry, Wm.
  milk sickness, 87
Jobling, J. W.
  spirochetal jaundice, 223
Jonati
  contagious abortion, 64
Jones, F. S.
  milk-borne scarlet fever epidemics, 267, 268
  scarlet fever streptococci, 267
Jones, T. H.
  scarlet fever in cows, 266
Jordan, E. O.
  animals responsible for food poisoning, 164
  aertrycke infection, 161
  Bacillus melitensis, 65
  distinguishing characteristics of aertrycke and schottmülleri, 161
  milk sickness, Bacillus lactimorbi, 87
  S. suipestifer in monkey, S. aertrycke in skunk, 165

K

Kalning
  mallein discovered by, 111
Kaneko
  spirochetal jaundice vaccination, 228
Kapadia
  plague, 201
Keefer, C. S.
  undulant fever, human infection, 80
Kelser, R. A.
  rabies, 128
  rabies virus, variation of virulence, 129
  vaccine against anthrax, 48
Kemmerer, T. W.
  plague without lesions, 199
Kennedy
  agglutination tests, abortus, 70
Kerlee, A. Leroy
  rocky mountain spotted fever, 241
Kiernan, J. A.
  tuberculosis, 13
  tuberculosis eradication of, 28
Kitasato, S.
  isolated plague bacillus, 194
  tetanus, 279
Klein, E.
  diphtheria in cats, 260
  diphtheria in cattle, 261
  scarlet fever in cows, 266
Klimmeck
  food poisoning in Prussia, 157
Knowlton, M.
  undulant fever, human infection, 80
Kober
  scarlet fever in cows, 267
Kober, G. N.
  tuberculosis, 11
Koch, Max
  avian tuberculosis, 22
  avian tuberculosis, 24
Koch, R.
  Clostridium oedematis-maligni, 286
  identification of anthrax bacillus, 40
  obtained pure culture of anthrax, 40
  tuberculosis, 2
  tubercle bacillus, tuberculin, 27
Kohn, L. A.
  S. enteriditis in cats, 165
Kolda, J.
  swine erysipeles, 171
Kolle
  vaccine against plague, 206

Koser, S. A.
  cause of food poisoning, 163
Krumwiede, C.
  cause of food poisoning, 163
  pestis-caviæ, 161
  S. enteriditis in cats, 165
  bovine mastitis, 254
  septic sore throat, 255
  tuberculosis, 13

L

Ladson, H. H.
  contagious abortion in bulls, 73
Laennec
  tuberculosis, 2
LaFosse
  "A Memoir of the Glanders of Horses, 110
Lamb, F. W.
  tularemia, 209, 211
Langenbeck
  actinomycosis, 94
Larson, W. P.
  contagious abortion, agglutinins and complement fixing bodies, 64
  tuberculosis, immunization against, 35
  tuberculosis immunization using carbon dioxide, 36
  undulant fever, complement fixation test, 79
Laveran
  rabies, 140
Law
  foot-and-mouth disease infection of from milk, 59
Lawrence
  contagious abortion in Europe, 63
Lawson
  plague, 201
Ledingham
  diphtheria in goats, 263
Leadingham, R. S.
  rat-bite fever, 232
League of Nations Health Organization
  plague mortality in India, 194
League of Nations Health Reports, 106
Lee, A. M.
  avian tuberculosis, 23
  septic sore throat, 256
Lehnert
  contagious abortion, 64
Leidy
  trichinosis, 175
Leiter, L. W.
  cause of food poisoning, 164
Levaditi, C.
  nature of the Negri bodies, 128
  the Negri bodies, 129
  vaccinia in brain of rabbits, 104
Linser
  swine erysipelas in man, 173
Lipschutz
  avian tuberculosis, 24
Lissner, L.
  ascaris infection in warmer climates, 181
Litterer
  abortus agglutination tests, 80
  cause of food poisoning, 163
  diphtheria in fowl, 261
Little
  scarlet fever streptococci, 267
Loeff
  smallpox, 102
Loeffler
  "Bacillus des Schweinenrotlaufs," 170
  foot-and-mouth disease filterable viruses, 57
  glanders, 111
Lord Macaulay
  smallpox, 100
von Lote
  rabies in frogs, 138
Lowden
  rabies, protozoan theory, named it, "Neurocytes hydrophobiæ," 127
Lowenstein
  avian tuberculosis, 24
Lyon, B. M.
  vaccination of dogs against rabies, 145
Lyons Commission
  smallpox in horses, 104
  vaccinia from smallpox, 103

M

McAlpine, J. G.
  abortus agglutination tests, 80
  undulant fever in man, 297
  undulant fever, porcine origine, 82
McCoy, G. W.
  Bacterium tularense, 209
  plague, 201

plague in ground squirrels, 200
tetanus, 280
tetanus organisms on scarifiers, 107
tularemia, 209, 214
vaccination against plague, 206

McClintic, T. B.
rocky mountain spotted fever, 240, 241

MacFadyean
avian tuberculosis, 23
contagious abortion test for, 64
contagious abortion in foxes, 75
glanders, agglutination test, 116
swine erysipelas, 170

McGavran, E. G.
fish tapeworm, 184

McKendrick
anti-rabic vaccination, 131
rabies, 141

McWeeney, E. G.
enteriditis infection, 160

Maffucci
tuberculosis, 3

Magath, T. B.
fish tapeworm, 184

Magendie
identified human rabies with disease in dog, 119

Maggiulli
rabies, 140

Mann, T. A.
septic sore throat, 254

Manson-Bahr, P. H.
plague, 302

March
glanders, tests for, 116

Marchaux, E.
anti-anthrax serum, 50

Marie
rabies, 139
rabies in frogs, 138

Marsh, H.
tuberculosis in swine, 26, 271

Martin, A.
spirochetal jaundice, 226

Marston
"Mediterranean remittent fever," 63

Mather
milk sickness, 86

Maver
rocky mountain spotted fever, 240

Maxey, E. E.
rocky mountain spotted fever, 236

Mayer
food poisoning in Germany due to B. enteriditis, 157

Mease
tularemia, 214

Mejio
anti-rabic treatment, 141

Metchnikoff
diphtheria in alligators, 264

Metropolitan Life Insurance Co.
rabies in children, 139

Meyer, K. F.
animals responsible for food poisoning, 165
botulism, 276
botulinus organism on apricots, cherries, and olives, 276
botulism, 276
Brucella abortus, 65
Brucella melitensis, 65
enteriditis in rats, 160
sporotrichosis in horses, 295
tetanus in man, 280

Mickle, F. L.
abortus agglutination tests, 80

Middleton
rat-bite fever, 232

Miller, J. L.
septic sore throat, 254

Minett, F. C.
diphtheria in horses, 263

Ministry of Health of Great Britain
plague in India, 194

Mitchell
tuberculosis, 13

Mitchell, O. W. H.
typhoid fever in chickens, 248

Miyajima
spirochetal jaundice, 223

Miyake, H.
rat-bite fever, 231

Mohler, J. R.
avian tuberculosis, 22
communicable diseases of cattle, 294
contagious abortion isolated organism from a child, 64
contagious abortion relation to human infection, 79
foot-and-mouth disease mode of infection, 59

foot-and-mouth disease from cowpox virus, 54
foot-and-mouth disease epidemic of in Central Europe 1911, 53
glanders, diagnosis of, 115
malta fever, 72
rabies virus transmitted through milk, 149
tuberculosis, 11
Mollerau
streptococcus infection, 250
Monti
spirochetal jaundice, 226
Moore, V. A.
undulant fever, human infection, 80
Morgagni
rabies infection, 119
Mori, N.
S. enteriditis in cats, 165
Morris, H.
anthrax spores, 42
Moscall
contagious abortion in Europe, 63
Moseley, E. L.
milk sickness, 87
Moses
food poisoning, 156
trichinosis in swine, 175
Munce
malta fever, 73

N

Nedrigailoff
rabies, 140
Nègre
parmelitensis, 66
Negri
rabies, diagnosis of, 120
Nevin
botulism in man, 277
Neil, M. H.
spirochetal jaundice, 223
Newman, G.
septic sore throat, 254
New Jersey State Dept. of Public Health
scarlet fever in cows, 267
New York City Health Dept.
anti-rabic treatment, 141
production of vaccine virus, 105
Nicolau, S.
nature of the Negri bodies, 128
vaccinia in brain of rabbits, 104
Nicolaier
tetanus, 279
Ninni, C.
tetanus, 279
Nixon, J. H.
rat-bite fever, 232
Noble, W.
tetanus in horses, 279, 280
Nocard, E.
contagious abortion, 64
Bacillus psittacosis, 152
rabies virus in milk and meat, 149
streptococcus infection, 250
tuberculosis, 7
Noguchi, H.
Leptospira, 222
spirochetal jaundice, 223
spirochetal jaundice, test for, 224
spirochetal jaundice vaccination, 228
rabies, protozoan organisms, 128
smallpox, 102
Nordholz, W. C.
glanders in man, 114
North, C. E.
septic sore throat, 255
Nuttall, G. H. F.
discovered, Clostridium welchii, 286
Nye, R. N.
vaccine virus, 102

O

Ofenheim
micrococcus tetragenous, 232
Ogata
Sporozoa muris, 232
Okudo
spirochetal jaundice vaccination, 228
Olitsky, P. K.
foot-and-mouth disease in guinea pigs, 56
foot-and-mouth disease in horse, 56
foot-and-mouth disease resistance to chemical agents, 57
Osborn, S. H.
anthrax, 42

Osiander
  glanders, transmissible to man, 110
Osler
  trichinosis in man, 178
Ost
  psittacosis, 152
Ostertag
  tuberculosis, 10
Ostrijanin
  rabies, 140
Otteraaen, A.
  spirochetal jaundice, 223
Ovid
  food poisoning, 156
Owen
  trichimosis, 175

P

Packard, J.
  rat-bite fever, 231
Pagniez, P.
  spirochetal jaundice, 224
Palamidessa
  Bacillus psittacosis, 152
Papon
  plague, 191
Park, W. H.
  glanders in man, 114
  rabies, 118, 140
  smallpox in monkeys, 104
  streptococci strains, 258
  tetanus, 279
  thermal death points of tubercle bacilli, table of 31, 32
  tuberculosis, 6, 13
Parker, R. R.
  rocky mountain spotted fever, 239
  tularemia, 214
  vaccination against rocky mountain spotted fever, 242
Parker, F.
  vaccine virus, 102
Parron, T.
  diphtheria from cattle, 262
  scarlet fever, 266
  septic sore throat, 255
Paschen, E.
  smallpox, 102
Patterson
  actinomycosis, 96
Pasteur
  anthrax in birds, 42
  anthrax vaccine, 40
  Clostridium oedematis-maligni, 286
  germ theory of disease, 2
  rabies, 119
  vaccine against anthrax, 48
  vaccination against rabies, 120
Pasteur Institute, Buenos Aires
  antirabic treatment, 141
Pasteur Institute in Paris
  vaccination against rabies, 129, 130, 131, 132, 139
Paul, G.
  smallpox, 102
Peacock
  trichinosis, 175
Pearse, R. A.
  tularemia, 211
Percival
  glanders, 110
Perroncito
  rabies virus in milk and meat, 149
Perry, J. C.
  tularemia, 214
Petit, L.
  spirochetal jaundice, 226
Pettit
  diphtheria in eels, 264
Petroff, S. A.
  tuberculosis, 36
Pfaff, F.
  swine erysipelas in poultry, 172
Pfeiffer, L.
  smallpox, 102
Philistines
  mortality in plague, 300
Pidoux
  tuberculosis, 2
Pirie, J. H. H.
  plague in rodents, 201
Pitt, W.
  swine erysipelas, 172
Pizzini
  tetanus in man, 279
Plutarch
  rabies, 118
Poels
  swine erysipelas in sheep, 171
  swine erysipelas in pigeon and fowl, 172
Pollender
  anthrax bacillus, 40
Power, W. H.
  scarlet fever in cows, 266

Preisg
  Corynebacterium abortus endemici, 65
Price-Jones, C.
  animals responsible for food poisoning, 165
Proescher, F.
  rat-bite fever, 231, 232
Procopius
  plague, 191
Provost
  cause of food poisoning, 163
von Prowazek, S.
  smallpox, 102

R

Rabinowitsch, L.
  avian tuberculosis, 22, 24
  tuberculosis in cattle, 6
  tuberculosis in cats, 8
Raffensperger, H. B.
  prevention of trichinosis, 179
Rahe, A. H.
  S. enteriditis in dogs, 165
Rahmel
  tetanus in man, 280
Ransom, H. B.
  mortality in trichinosis, 178
  resistance of trichinæ, 180
  trichinosis in man, 177
Ravenel, M. P.
  tuberculosis, 11
Rayer
  anthrax bacillus, 40
  glanders, 111
Raynaud
  paramelitensis, 66
Read, W. J.
  enteriditis in rats, 160
Regan, J. C.
  anthrax vaccination in Argentine, 50
  anthrax serum treatment, 50
Reece, M.
  rat-bite fever, 231
Reichel, J.
  incubation period of rabies in domestic animals, 135, 136
  vaccination of dogs against rabies, 146
Reid
  tuberculosis, 2
Reiter, H.
  spirochetal jaundice, 225
  Spirocheta nodosa, 222
Renault
  glanders, 111
Rettger, L. F.
  Salmonella pullorum, 162
  tetanus organism in gelatin, 280
  white diarrhea, 302
Reynolds, F. H. K.
  malta fever, 72
Rhoades, B.
  tularemia, 214, 216
Rice, T. B.
  deaths from rabies, 138
  statistics on world prevelance of rabies, 124
Ricketts, H. T.
  causative agent of rocky mountain spotted fever, 238, 239, 240
Ritter, F.
  psittacosis, 152
de Rivas, Damaso
  ascaris infection in man, 181
Rivolta
  tuberculosis, 3, 20
Robertson
  plague, 204
  plague in hot climates, 195
Robins, A. O.
  cure of glanders with mallein, 114
  glanders in man, 113, 114
Robinson, G. H.
  spirochetal jaundice, 223, 224
Rogers, O. F.
  spirochetal jaundice, 226
Rosenau, M. J.
  cause of food poisoning, 163
  cowpox, 103
  foot-and-mouth disease from contaminated vaccine virus, 54
  tetanus in vaccine, 107
Roux
  diphtheria in sheep, 263
  rabies, 140
Ruini
  glanders, 110
Ruprecht
  swine erysipelas in man, 173
Russell, J. B.
  scarlet fever in cows, 266

Rühling
  tuberculosis, 2
Ryle, J. A.
  spirochetal jaundice, 223, 225

S

Salmon
  actinomycosis, 96
  suipestifer infection, 159
Salthe
  cause of food poisoning, 163
  pestis-caviæ, 161
Sanford
  actinomycosis, 95, 96, 97
  actinomycosis in males, 96
  actinomycosis in U. S.—table of, 98
Sattler, R.
  tularemia, 211
Savage, W. G.
  aertrycke infection, 161
  B. proteus, 159
  cause of food poisoning, 162, 163
  diphtheria in cats, 260
  enteriditis infection, 160
  enteriditis in rats, 160
  food poisoning, predisposing factors, 165, 166
  food poisoning in Britain, 157
  mammitis, 253
  salmonella group—table of, 166
  salmonella organism in food poisoning—table of, 160.
  suipestifer infection, 159
Schern
  vaccination of dogs against rabies, 146
Schipp, C.
  swine erysipelas in chickens, 171
Schlesinger, M. J.
  rocky mountain spotted fever, 239
Schlingman, A. D.
  vaccination of dogs against rabies, 146
Schlotthauer, C. F.
  mastitis, 253
Schneider
  vaccination of dogs against rabies, 146
Schnurer
  vaccination of dogs against rabies, 146
Schoen, R.
  nature of the Negri bodies, 128
Schoening, H. W.
  "street virus" distinct from other viruses, 129
  vaccination against rabies, 146
School of Wound Bacteriology
  gas gangrene, 285
Schottmüller
  rat-bite fever, 233
  Streptothrix muris ratti, 232
Schroeder, E. C.
  contagious abortion, 64
  contagious abortion in sheep and goats, 75
  tuberculosis, 36
  undulant fever, 79
Schubert
  glanders, complement fixation test, 116
Sclavo
  anthrax, 50
Scott, J. M.
  smallpox, 102
Schütze, H.
  aertrycke group, 161
  glanders, 111
  glanders, complement fixation test, 116
Schwartz
  prevention of trichinosis, 179
Schwarze
  botulism in cattle, 276
Sedgwick, J. P.
  contagious abortion, agglutinins and complement fixing bodies, 64
  undulant fever, complement fixation test, 79
Sellards, A. W.
  spirochetal jaundice, 224
Sellers, T. F.
  deaths from rabies, 138, 139
Selter
  psiltacosis, 152
Semple
  anti-rabic vaccination, 133
Semple—malta fever
  agglutination test for, 63
Sequin, P.
  discovered Clostridium fallax, 287
  Clostridium oedematiens, 287
  gas gangrene, 285

Welch bacillus infection, 286
Shaw
  Brucella abortus, 65
  Brucella melitensis, 65
  Streptococcus epidemicus in milk, 252
Siebold
  trichinosis, 175
Siegel
  foot-and-mouth disease, mortality of in man, 59
Simond
  plague, 202
Simon, G.
  anti-rabic treatment, 141
Simon, C. E.
  smallpox, 102
Simmons, J. S.
  diphtheria in cats, 260, 299
Simson
  plague, 201
Sivori-Rebaudi
  rabies, 140
Skellet
  contagious abortion in Europe, 64
Slanetz, C. A.
  undulant fever, porcine origin, 82
Smith, N.
  swine erysipelas in man, 173
Smith, E. R.
  elimination of tuberculosis in poultry, 29
Smith, W. V.
  contagious abortion in swine, 75
Smith, Theobald
  abortion in swine, 67
  bovine mastitis, 254
  hemolytic streptococci alpha and beta types, 251
  septic sore throat, 255
  streptococcus, 250
  suipestifer infection, 199
  swine erysipelas, 170
  tuberculosis, 3
Smyth, H. F.
  anthrax, 43
  anthrax—human sources of infection, table of 47
  anthrax, disinfecting hides with iodine, 49
  anthrax—human, table of, 44
Society of American Bacteriologists
  alcaligines, 66
  Bacterium abortus, 65
  Bacterium melitensis, 65
Sobernheim
  vaccine against anthrax, 48
Solleysel
  glanders, 110
Songkla, M.
  fish tapeworm in man, 184
Spencer, R. R.
  rocky mountain spotted fever, 239
  vaccination against rocky mountain spotted fever, 242
Spinola
  tuberculosis, 2
Spray, R. S.
  cause of food poisoning, 163
  enteriditis infection, 161
State Department of Agriculture of Illinois
  milk sickness, Experimental feeding of white snake root, 92
  pasteurization, 26
Stewart
  cause of food poisoning, 163
Stewart, A. V.
  tularemia, 217
St. Cyr
  contagious abortion, 64
Stiles
  prevention of trichinosis, 180
Stimson, A. M.
  rabies, 137
  rabies, seasonal prevalence of, 121
Stitt
  malta fever, 78
Stockman
  contagious abortion test for, 64
  contagious abortion in foxes, 75
Stokes
  actinomycosis, 94, 96
  actimonycosis in children, 96
Stokes, A.
  spirochetal jaundice, 223, 225
Storrs Experiment Station
  malta fever, 73
Stribolt
  discovered Bacillus abortus, 64
Strong, R. P.
  vaccine against plague, 206
Swithinbank, H.
  septic sore throat, 254

Symmers—anthrax
  with Eichham's serum, 50
Symmers, D.
  spirochetal jaundice, 226

T

Taylor, K.
  gas gangrene, 287
Teage
  vaccination against plague, 206
Teideman
  trichinosis, 175
Tenker
  trichinosis, 175
Ten Broeck, C.
  swine erysipelas, 170, 172
  tetanus, 283, 280
Theiler, M.
  spirochetal jaundice, 224
Thompson
  malta fever, 72
Thomas, R. C.
  streptococcus in milk, 252
Titze
  avian tuberculosis, 17
Todd, C.
  diphtheria in cattle, 261
Tonney, F. O.
  tubercle bacilli in milk, 17
Torrey, J. C.
  S. enteriditis in dogs, 165
Torstenson, H.
  malta fever, 70
Toussant, H.
  anthrax—serum cure, 49
Towler, H. H.
  spirochetal jaundice, 225
Trask, J. W.
  diphtheria from cattle, 262
  scarlet fever, 266
Traum, J.
  contagious abortion, relation to human infection, 79
  contagious abortion, isolated organism from a child, 64
  contagious abortion in swine, 75
Travers
  glanders, 111
Tulloch, W. J.
  tetanus in man, 280
Tunnicliff, R.
  rat-bite fever, 233
Tyson
  undulant fever caprine origin, 78
Tytler, W. H.
  spirochetal jaundice, 223

U

Uhlenhuth
  animals responsible for food poisoning, 164
Umeno
  vaccination of dogs against rabies, 145
U. S. Census Bureau
  food poisoning, 157
U. S. Department of Agriculture
  malta fever, 72
  rocky mountain spotted fever, 242
U. S. Public Health Service
  importation of hair and hides, 48
  diphtheria from cattle, 262
  plague, 203, 207
  production of vaccine virus, 105
  recent methods of vaccination, 108
  rocky mountain spotted fever, 238, 242
  septic sore throat, 255
  septic sore throat epidemics, 257

V

Vail
  tularemia, 211
Valentine
  bovine mastitis, 254
  S. enteriditis in cats, 165
  septic sore throat, 255
Valery-Radot
  tuberculosis, 2
Van der Loeff, A.
  smallpox, 102
Van der Reis, V.
  tetanus in man, 280
Van Es
  tubercle bacilli, from various sources, 16
Van Swieten
  rabies in man, 119
Van Velzen
  swine erysipelas, 172
Vegetius
  glanders, first systematic description of, 110

Verder, E.
  aertrycke infection, 161
  enteriditis in rats, 160
Vergeer, T.
  fish tapeworm in dogs, 184
Viborg, Eric
  glanders and farcy identical, 110
Villemin
  tuberculosis, 2
Virchow
  life cycle of trichina, 175
Vitet
  glanders, 110
Voelker
  actinomycosis, 95
  actinomycosis in U.S.—table of, 98
Voight, L.
  smallpox in sheep, 104
  vaccinia from smallpox, 103
Volpi
  glanders, 110

W

Wade
  tularemia, 214
Wadsworth, A. B.
  spirochetal jaundice, 223, 226
Wagner
  psittacosis, 152
Walch, E. W.
  spirochetal jaundice, 223
Walker, J. E.
  spirochetal jaundice, 225
Wall
  contagious abortion test for, 64
Walsh, W. E.
  milk sickness, 87, 92
Wani
  spirochetal jaundice, 223
Wang, C. K.
  tuberculosis, 13
Waterhouse
  vaccination in U. S., 101
Warthin, A. S.
  fish tapeworm in man, 184
Washburn, H. J.
  anthrax—description of the disease, 43
  avian tuberculosis, 22
Watson, E. A.
  avian tuberculosis, 23
  tuberculosis, 36
  glanders, vaccination against, 115
Watson, J.
  rat-bite fever, 231
Weaver
  scarlet fever in monkeys, 269
Weber
  avian tuberculosis, 24
Weichel
  animals responsible for food poisoning, 165
Weinberg, M.
  discovered Clostridium fallax, 287
  Clostridium oedematiens, 287
Welch, W. H.
  diphtheria in cats, 260
  discovered Clostridium welchii, 286
  gas gangrene, 285
  welch bacillus infection, 286
Wells
  tetanus, 281
Weiss
  cause of food poisoning, 163
Wernicke
  diphtheria in sheep, 263
Wherry, W. B.
  plague in ground squirrels, 200
  tularemia, 211
White, P. B.
  aertrycke infection, 161
  cause of food poisoning, 162, 163
  enteriditis in rats, 160
  food poisoning in Britain, 157
  food poisoning, predisposing factors, 165
  suipestifer infection, 159
White, B.
  deaths from smallpox, 106
  glanders, vaccination against, 115
  septic sort throat, 255
White, Norman
  plague, 204
White, J. L.
  tubercle bacilli in milk, 17
Whitmore
  rabies, 118
Wibert
  epidemic in chimpanzees, 302
Wilcox, W.
  rat-bite fever, 231
Williams, A. W.
  rabies, protozoan theory, named it "Neurocytes hydrophobiæ," 127

Williams, C. L.
  lesions in plague, 199
  plague without lesions, 199
Williams, H. U.
  trichinosis in man, 178
Williams, R. S.
  tubercle bacilli in cow manure, 17
Wilson
  rabies, vaccination against, 120
Wilson, L. B.
  rocky mountain spotted fever, 236, 238, 240
Winslow, C. E. A.
  pestis-caviæ, 161
  septic sore throat, 254
  tuberculosis mortality, 31
Wladimiroff
  diphtheria in cattle, 261
  diphtheria in goats, 263
Wolbach, S. B.
  Dermacentroxenus rickettsi, 239
  rocky mountain spotted fever, 239
Wolf
  actinomycosis, 94
Wood, M. W.
  rocky mountain spotted fever, 236
Wooton, E. O.
  milk sickness, 87
Wright
  actinomycosis, 96
Wright—malta fever
  agglutination test for, 63
Wu Lien-Teh
  control of plague, 201
  plague, 197
Wyrsykowski
  rabies virus, action on gastric juice, 149

Y

Yabe, S.
  diphtheria in cats, 260
Yersin
  diphtheria in sheep, 263
  isolated plague bacillus, 194
  vaccine against plague, 206

Z

Zeulzer
  spirochetal jaundice, 226
Zinke
  rabies-virulence of saliva from a dog, 119
Zwick
  animals responsible for food poisoning, 165
  contagious abortion test for, 64
Zwinger
  rabies infection, 119

INDEX; SUBJECT

INDEX OF SUBJECTS

A

Abortus bacillus
  in man, 76
  in milk, 79
  virulence of, 75
Abortus-melitensis group, 83
Abortus organism, characteristics of, 66
Actinomyces organism, characteristics of, 95
Actinomycosis, 94
  in animals, 94, 96
  animals susceptible to, 94
  in cats, 96
  in cattle, 94, 96, 294
  in cows, *see* cattle
  in dogs, 94, 96
  in geese, 94, 96
  in goats, 94
  in guinea pigs, 94
  in hogs, *see* swine
  in horses, 94, 96, 296
  in lizards, 94, 96
  in man, 94, 96, 97
  in mice, 94
  in monkeys, 94, 96
  in rabbits, 94
  in sheep, 94, 96
  in swine, 94, 96, 297
  characteristics, cultural, 94
  characteristics, pathogenic, 94
  confused with, 97
  history of, 94
  geographical distribution, 95
  prevention of, 97
  symptoms of, 95
  types of organisms, 94
   Actinomyces actinoides, 95
    Actinomyces asteroides, 95
    Actinomyces bovis, 94
    Actinomyces candidus, 95
    Actinomyces farcinicus, 95
    Actinomyces freeri, 95
    Actinomyces gedanensis, 95
    Actinomyces hominis, 94
    Actinomyces maduræ, 94
    Actinomyces necrophorus, 95
   in U.S., table of
Acute hemorrhagic jaundice, *see* spirochetal jaundice
Aertrycke group
   Arkansas type, 161
   Binus type, 161
   C. type, 161
   Hirschfield type, 161
   "Mutton" type, 161
   Newport type, 161
   Stanley type, 161
Aertrycke infection, 161
  in animals, 161
  in cattle, 161
  in guinea pigs, 161
  in man, 161
  in mice, 161
  in rabbits, 161
  in rats, 161
  in sheep, 161
  in swine, 161
Aertrycke and Schottmülleri
  distinguishing characteristics of, 161
Agglutination test
  for glanders, 116
"Alcaligines," *see* Brucella, 66
Alcaligines abortus
  contagious abortion, 65
Alcaligines melitensis
  malta fever, 65
Alexandrian rat
  plague, 198
Alkali disease, *see* milk sickness
Alligators,
  diphtheria in, 264
Ancients
  food poisoning in, 156
  plague in, 191
Animals susceptible to
  actinomycosis, 94
  anthrax in, 42
  botulism in, 275, 276
  contagious abortion in, 72
  foot-and-mouth disease in, 55, 58
  glanders in, 112
  malta fever in, 69
  diphtheria in, 260
  plague in, 195, 198
  rabies in, 134
  rat-bite fever in, 232
  rocky mountain spotted fever in, 240
  scarlet fever, 266-272
  septic sort throat in, 253
  swine erysipelas in, 171
  tetanus in, 249, 279

  trichinosis in, 175
  tuberculosis, 1, 37
  tuleremia, 212
  animal parasites, 175-187
Antelope
  foot-and-mouth disease, 55
Anthrax, 39
  in animals, 42
  in cattle, 42, 294
  in deer, 302
  in dogs, 42, 298
  in fowls, 42
  in goats, 42, 297
  in guinea pigs, 42
  in horses, 42, 296
  in man, 44
  in mice, 42
  in rabbits, 42
  in sheep, 42
  in swine, 42, 297
  anti-anthrax serum, 50
  carriers of, 42-43
  in China, 47
  in dead animals, 49
  deaths from, in U. S. by occupation, from 1910-17, table of, 46
  disinfecting stations in England, 49
  disposing of dead animals, 49
  Eichhorn's serum, 50
  in fields, 49
  from shaving brushes
    in the British army, 48
    in Illinois, 48
  history of, 39
  horse hair, 47
  infection, 42
  mortality, report on, by British ministry of health, 50
  occupational, 45, 47
  other names for it, 39
  prevalence of, 41
  prevention and control, 48
  regulations of importation of hair and hides in U. S., 48
  serum cure, 49
  serum vaccination in Argentine, 50
  shaving brushes, 47
  in Siberia, 47
  sources of infection, 42-48
  sterilization of hides, hair, and wool, 49
  tanning industry, 47
  vaccination against, 48
Anthrax bacillus, 40
  resistance of, 41
Anthrax, human
  records of fatal cases in U. S. from 1910-17, 44
Anthrax vaccine, 40
Anti-rabic treatment, 141
  of dogs, compulsory in, 147
  methods of, 141
Anti-rabic vaccination, 141
  failures of, 146
  table of, 130-33
Antitoxin
  against botulism, 277
  against tetanus, 281
Apes
  tuberculosis, 6
  tuberculosis, human, 271
Aphthous fever, *see* foot-and-mouth disease
Aplopappus heterophyllus, *see* rayless goldenrod
Arctomys bobac, *see* tarbagan
Ascaris infection
  in animals 181
  in children, 181
  in man, 181, 185, 186
  in swine 181
  prevention of, 181
  in warmer climates, 181
Ascaris lumbricoides, 181, 186, 301
Ascaris suis
  in swine, 181
Ass
  cowpox in, 109
  rabies in, 134

B

Baboons
  Samonelle aertrycke, 302
Bacillus of hog cholera, 159
Bacillus abortus
  contagious abortion, 64, 65
  in cows milk, 65
  undulant fever, 79
Bacillus aerogenes capsulatus, *see* Clostridium welchii
Bacillus aertrycke, 158, 161
Bacillus aertrycke, *see* Salmonella aertrycke
Bacillus cholerae suus, 158, 159

Bacillus cholerae suus, *see* food poisoning
Bacillus cholerae suum, 159
Bacillus coli, 253
Bacillus enteriditis Gaertner, *see* food poisoning
Bacillus enteriditis sporgenes, *see* Clostridium welchii
Bacillus erysipelatus suis, *see* swine erysipelas
Bacillus of Ghon, 286
Bacillus lactimorbi, 87
Bacillus lactis aerogenes, 253
Bacillus mallei, *see* Pfeifferella mallie, 111
Bacillus melitensis, malta fever, 65
Bacillus murisepticus, *see* septicemia in mice
Bacillus neigeux, *see* Clostridium oedematiens
Bacillus oedematis maligni Koch, *see* Clostridium oedematis-maligni
Bacillus perfringens, *see* Clostridium welchii
Bacillus pestis
  plague, 194
Bacillus pestis-caviae, 158
Bacillus phlegmonis emphysematosæ, *see* Clostridium welchii
Bacillus psittacosis, 152
Bacillus pyogenes, 253
Bacillus rhusiopathiae suis, *see* swine erysipelas
Bacillus of Sachs, 286
Bacillus sanguinarium, *see* food poisoning
Bacillus welchii, *see* Clostridium welchii
Bacillus of van Hibler 286
Bacterium abortus
  contagious abortion, 65
Bacillus enteriditis Gaertner, 157, 158, 159
  food poisoning, 157
Bacillus sanguinarium, 158
Bacillus suipestifer, 157
  food poisoning, 157
Bacillus suipestifer-voldagsen
B. suipestifer-voldagsen
Bacillus typhi-suis
  in swine, 164
B. C. G. organism, 35
Bact. abortus, 80
  see Brucella abortus
Bacterium psittacosis, *see* Salmonella psittacosis
Bacterium suipestifer, *see* food poisoning, 157, 158, 159
Bacterium tularense, 209, 211, 213, 217
  characteristics of, 213
Badger
  trichinosis in, 177
  rocky mountain spotted fever in, 241
  rocky mountain spotted fever, 302
Bats
  plague in, 195, 201
Bear
  rocky mountain spotted fever, 302
  trichinosis in, 177
Bed bug, 215
Beef tapeworm, 182, 186
  in cattle, 294
Bilious remittant fever, *see* Malta fever
Birds
  botulism in, 276
  rabies in, 137
  tuberculosis, 6, 8
Bison
  foot-and-mouth disease, 55
"Black bain," *see* anthrax
"Black Death," *see* plague, 192
Black Fever, *see* Rocky Mountain Spotted Fever
Blackleg
  in cattle, 294
Black tongue, *see* rabies, 118
Blue Disease, *see* Rocky Mountain Spotted Fever
Booby owl,
  plague in, 201
Botulinus bacillus, 156
Botulism, 156, 249, 275
  in animals, 275, 276
  in birds, 276
  in cats, 277
  in cattle, 276, 294
  in chickens, 275
  in dogs, 276
  in fishes, 277
  in frogs, 277
  in goats, 276
  in horses, 276
  in man, 275, 276, 277

- in mice, 277
- in poultry, 276
- in rabbits, 277
- in rats, white, 277
- in swine, 276
- antitoxin against, 277
- in cheese, 277
- in corn, 277
- in food, 277
- in olives, 277
- prevention of, 277
- source of infection, 275, 276
- in spinach, 277
- in string beans, 277

Boneset, *see* white snake soot
Bovine mastitis, 253
Brucella designated Br., 66
Brucella
- malta fever, 66

Brucella abortus, 83, 295, 297
- affinity for, 73
- contagious abortion, 62, 65
- destruction of, 82
- in horses, 296
- in man, 76
- virulence of, 75

Brucella melitensis, 83, 297, 299
- malta fever, 62, 65
- in goats, 297
- in horses, 296
- in man, 76

Brucella organism
- characteristics, 66

Bruce's septicemia, *see* malta fever
Buffalo
- foot-and mouth disease, 55

Bull Fever, *see* Rocky Mountain Spotted Fever
Bulls, contagious abortion in, 73, 75
Bubonic plague, *see* Plague
Butter bacillus, 5

C

California ground squirrel
- plague in, 302

Callospermophilus lateralis cinerassens, *see* Side-striped ground squirrel
Calves, *see* cattle
- avian tuberculosis, 23

Camel
- cowpox in, 104
- foot-and-mouth disease, 55
- plague in, 195, 201

Caprine, *see* goat
Carp, tuberculosis, 4
Cats
- actinomycosis in, 96
- botulism in, 277
- diphtheria in, 260, 261, 299
- dipylidium caninum in, 185
- foot-and-mouth disease, 56, 299
- flukes, 299
- glanders in, 112, 299
- malta fever, 70
- plague in, 195, 201, 299
- poliomyelitis in, 247
- rabies in, 134, 135, 137, 299
- rat-bite fever in, 231, 232, 299
- round worm in, 299
- S. enteriditis in, 165
- scarlet fever in, 269
- tetanus in, 281
- trembles in, 91
- trichinosis in, 177
- tuberculosis, 6, 8, 299
- tuberculosis, human, 271
- tularemia in, 213, 299
- undulant fever, 299
- vaccinia in, 104
- white diarrhea in, 162

Cattle
- actinomycosis in, 94, 96, 294
- aertrycke infection in, 161
- anthrax in, 42, 294
- beef tapeworm, 294
- blackleg, 294
- botulism in, 276, 294
- cattle fracy, 294
- contagious abortion in, 62, 294
- contagious pleuropneumonia, 294
- chronic bacterial dysentery, 294
- cowpox in, 103, 104, 294
- diphtheria in, 261, 262, 294
- dog tapeworm in, 185
- food poisoning in, 164
- foot-and-mouth disease in, 55, 294
- flukes in, 185
- gas gangrene in, 287, 294
- hemorrhagic septicemia, 294
- horsepox in, 104
- infectious ophthalmia, 294
- malignant catarrh, 294
- malignant edema, 294
- mammitis in, 253
- nagana in, 294
- necrotic stomatitis, 294
- plague in, 195, 201
- poliomyelitis in, 248

  rabies in, 134, 135, 137, 294
  rinderpest, 294
  rocky mountain spotted fever in, 241
  salmonella infection, 294
  scarlet fever in, 266, 269, 294
  septic sore throat in, 250, 253, 254, 255
  smallpox in, 271
  swine erysipelas in, 171
  tetanus in, 279, 280, 294
  Texas fever, 294
  trembles in, 90
  trichinosis in, 177
  tuberculosis, 6, 7, 294
  tuberculosis, human, 271
  tularemia in, 213
  typhoid fever in, 248
  white scours, 294
Cattle farcy, in cattle, 294
Causative agent, of food poisoning, 155, 162, 163
  of rat-bite fever, 232
  of rocky mountain spotted fever, 238
  of swine erysipelas, 170, 171
Ceratophyllus acutus, *see* squirrel flea
Ceratophyllus fasciatus, *see* fleas, 202
Ceratophyllus silvantieri, *see* fleas, 202
Chamois
  foot-and-mouth disease, 55
Characteristics
  of actinomyces organisms, 95
  of actinomycosis, 94
  of glanders bacillus, 111
Charbon, *see* anthrax
"Charbon Malin," *see* anthrax
Cheese
  botulism in, 277
Chicken, *see* also poultry
  botulism in, 275
  malta fever, 70
  plague in, 195, 201
  poliomyelitis in, 247, 248
  rabies in, 135
  S. gallinaum in, 165
  S. pullorum in, 165
  scarlet fever in, 269
  tetanus in, 281
  trichinosis in, 177
  tularemia in, 213
  typhoid fever in, 248
  white diarrhea in, 158, 162
Chicken-pox, 104
Children
  ascaris infection in, 181
  rabies in, 139
  scarlet fever in, 269
  Weil's disease, 302
Chipmunk
  tularemia in, 213
Chronic bacterial dysentery
  in cattle, 294
Chrysops discalis, 211
  tularemia, 212
Cimex lectularius, *see* bed bug
Citellus beecheyi, *see* ground squirrels
Citellus columbianus, *see* Columbian ground squirrel
Cockatoo
  tuberculosis, 6, 8
  tuberculosis, human, 271
Clostridium botulinum, 275
Clostridium falax, gas gangrene, 287
Clostridium oedematiens, gas gangrene, 287
Clostridium oedematis-maligni, 286
Clostridium tetani, 279
Clostridium welchii, gas gangrene, 285
Complement fixation test
  for glanders, 116
Common thoroughwort, *see* white snake root.
Communicable diseases
  list of, 247
Consumption (tuberculosis), 1
Contagion
  horses, 295
Control
  of glanders, 115
  of plague, 201
Contagious abortion, 62
  in animals, 72
  in bulls, 73, 75
  in cattle, 294
  in foxes, 75
  in goats, 75
  in hogs, map of, 81
  in man, 62, 76
  in sheep, 75
  in swine, 62, 75, 297
  confused with other diseases, 63
  control and prevention of, 75
  in Europe, 63, 64

  geographical prevalence, 67
  history of, 63
  relationship to human infection, 63, 79
  seasonal prevalence, 69
  symptoms of, 73
  test for, 64
Contagious abortion organism, 66
Contagious pleuropneumonia
  in cattle, 294
Contagious pneumonia, horses, 295
Continued fever, *see* undulant fever, 78
Cow, *see* cattle
  malta fever, 70
Cowpox, 103
  in animals
  in ass, 104
  in camels, 104
  in cattle, 103, 104, 294
  in goats, 104
  in horse, 104
  in monkeys, 104
  in sheep, 104
  in rabbits, 104
  etiological agent, 101
  history of, 100
Columbian ground squirrel
  rocky mountain spotted fever in, 240
Corn
  botulism in, 277
Ctenocephalus acutus, *see* fleas, 202
Ctenocephalus canis, *see* fleas, 202
Ctenocephalus felis, *see* fleas, 202
Ctenopsylla musculli, *see* fleas, 202
Corynebacterium abortus endemici
  contagious abortion, 65
Corynebacterium diphtheria, 260
Coyote
  tularemia in, 213, 217
  rocky mountain spotted fever, 302
Cyprus fever, *see* malta fever
Cytoryctes variola-vaccinæ, 102

D

Danubian fever, *see* malta fever
Deaths from smallpox, 106
Deer
  anthrax, 302
  foot-and-mouth disease, 302
Deer fly, 215
Deer-fly fever, *see* tularemia
Dermacentor, 302
Dermacentor, andersoni
  tularemia, 212
Destruction of rats, methods of, 205, 207
Dermacentroxenus rickettsi, 239
Dermacentor variabilis, *see* tick
Diagnosis
  of glanders, 115
  of rabies, 120
  of trichinosis, 179
Diphtheria, 260
  in animals, 260
  in alligators, 264
  in cats, 260, 261, 299
  in cattle, 261, 262, 294
  in dogs, 260, 263
  in domestic fowl, 261
  in eels, 264
  in frogs, 264
  in fowls, 260, 261
  in goats, 263
  in guinea pigs, 261, 263
  in horses, 261, 263
  in man, 260, 261, 262
  in mice, 263
  in monkeys, 263
  in poultry, 302
  in rabbits, 263
  in rats, 263
  in sheep, 263
Dipylidium caninum, *see* tapeworm
  in cats, 185
  in dogs, 185
Diphyllobothrium latum, 183, 186
  fish tapeworm, 181, 182, 186
Discovery of mallein, 111
Diseases confused with trichinosis, 179
Dogs, 297
  actinomycosis in, 94, 96
  anthrax in, 42, 298
  botulism in, 276
  diphtheria in, 260, 263
  dipylidium caninum in, 185
  fish tapeworm in, 184
  flukes in, 185
  foot-and-mouth disease, 56, 298
  gas gangrene in, 287
  glanders in, 112, 298
  guinea worm, 298
  malta fever, 70

  mange, 298
  plague in, 195, 201
  poliomyelitis, 247, 248
  rabies in, 134, 135, 136, 137, 298
  rat-bite fever in, 233, 299
  ringworm, 298
  S. enteriditis in, 165
  scarlet fever in, 269, 299
  spirochetal jaundice in, 225
  tapeworm in, 184, 186, 298
  tetanus in, 281
  trembles in, 91
  trichinosis in, 177, 299
  tuberculosis, 6, 8, 298
  tuberculosis, human, 271
  tularemia in, 213
  compulsory anti-rabic treatment of, 147
Dog bites, treatment of, 142
Dog-pox, 104
Dog, rabies
  symptoms
    dumb or paralytic rabies, 136
  furious rabies, 136
  vaccination of against rabies, 146
Dog tapeworm, 184, 186
  in animals
  in cattle, 185
  in man, 184
  in sheep, 185, 297
  taenia echinococcus, 184, 186
Domestic fowl, diphtheria in, 261
Donkeys
  glanders in, 112
  plague in, 195, 201
  tuberculosis, 8
Dourine in horses, 295
Dracunculus medinensis, *see* guinea worm
Ducks, *see also* poultry
  malta fever, 70
  S. anatum in, 165
Dust fever, *see* malta fever
Dwarf tapeworms
  Hymenolepsis diminuta, 301
  Hymenolepsis nana, 301

E

Earthworm
  as anthrax carrier, 49
Eels
  diphtheria in, 264
Eel worm, *see* Ascaris lumbricoides
Eggs, tubercle bacilli in, 22
Elimination of rabies in dogs, 143
English rat, plague, 198
Enteriditis group
  in food poisoning, 158
  in animals
  in man, 160
  in rats, 160
  infection, 159, 160
  bovine origin, 160
  porcine origin, 160
  source of infection, 160
Epidemic jaundice, *see* spirochetal jaundice
Epidemic of undulant fever, 78
Epithelioma contagiosum in fowl, 204
Epizoötic aphtha, *see* foot-and-mouth disease
Equine, *see* horse
  contagious abortion, 66
Erysipelothrix murisepticæ, *see* septicemia in mice
Erysipelothrix rhusiopathiæ, *see* swine erysipelas
Etiological agent
  of cowpox, 101
  of smallpox, 101
Eupatorium serotinum, *see* white snake root
Eupatorium urticæfolium, *see* white snake root
Eutamias luteiventris, *see* yellow bellied chipmunk
Eutamias quadrivittatus umbrinus, *see* white bellied chipmunk
Experimental feeding of white snake root, 91
Exzema contagiosa, *see* foot-and-mouth disease

F

"Farcy," *see* glanders, 112
Farcy buds, 113
Fasciola hepatica, 297
Febris melitensis, *see* malta fever
Febris undulans, *see* malta fever
Ferrets
  glanders in, 112
  plague in, 201
  rat-bite fever in, 231, 233

Fish
  botulism in, 277
  flukes in, 185
  tapeworm in, 182, 183, 184, 186
  tuberculosis, 4
Fish tapeworm, (Diphyllobothrium latum), 181, 182, 183, 186
  in dogs, 184
  in man, 184, 186
"Fixed virus," *see* rabies
Fleas, 202
  plague in, 202
Flukes
  in animals, 185
  in cats, 299
  in cattle, 185
  in dogs, 185
  in fish, 185
  in man, 185
  in sheep, 185, 297
  in snail, 185
  Clonorchis sinensis, 185
  Fasciolopsis, 185
  Fasciola hepatica, 185
  Opisthorchis felineus, 185, 299
  Opisthorchis pseudofelineus, 299
  Paramphistomum cervi, 185
Food, botulism in, 277
Food Poisoning, 155
  aertrycke infection, 160, 161
  in the ancients, 156
  animals responsible for, 164, 165
  in cattle, 164
  in sheep, 164
  in swine, 164
  Bacillus enteriditis, 157, 159, 160
  Bacillus pestis-caviæ, 158
  Bacillus proteus, 159
  Bacillus suipestifer, 157
  causative agent of, 155
  cause of, 162, 163
  enteriditis infection 159, 160
  "fowl typhoid," 158, 162
  groups
    enteriditis group, 158
    Gaertner group, 155, 157, 158
    hog cholera group, 158
    intermediate group, 158
    salmonella group, 155, 157, 158
  history of, 156
  predisposing factors, 165
  prevalence of, 156, 166
  organisms of,
    Salmonella abortus-equi, 158, 162
    Salmonella aertrycke, 158
    Salmonella anatum, 158
    Salmonella enteriditis, 158, 159
    Salmonella gallinarum, 158
    Salmonella paratyphi, 158
    Salmonella pestis-caviæ, 158, 161
    Salmonella psittacosis, 158
    Salmonella pullorum, 158
    Salmonella schottmülleri, 158
    Salmonella suipestifer, 158, 159
  suipestifer infection, 159, 160
Foot-and-mouth diseases, 53
  animals susceptible, 55
  in animals, 58
  in antelope, 55
  in bison, 55
  in buffalo, 55
  in camels, 55
  in cats, 56, 299
  in cattle, 55, 294
  in chamois, 55
  in deer, 302
  in dogs, 56, 298
  in giraffe, 55
  in goats, 55, 297
  in guinea pigs, 56
  in horse, 56, 296
  in llama, 55
  in man, 59
  in poultry, 56
  in rabbits, 56
  in rats, 56
  in sheep, 55, 297
  in swine, 55, 297
  "border war" against, 54
  control of vaccine, 54
  cultivation of organism, 57
  death rate from, 58
  description of, 58
  epidemics of, 53
  from contaminated vaccine virus, 54
  from cowpox virus, 54
  geographical prevalence, 55
  history of, 53
  human carriers of, 59
  incubation period, 58
  infectious agent, 57
  infection of from milk, 59
  malignancy of, 58
  mode of infection, 58, 59
  mode of infection-table of, 61
  mortality of in man, 59
  prevention of in cattle, 60
  prevention of in man, 60

  resistance to, in man, 60
  resistance of the virus to chemical agents, 57
  resistance of the virus to temperature, 57
  serious epidemic of 1914, 54
  similarity to organism causing vesicular stomatitis in horses, 57
  symptoms of, 58, 59
  vesicular stomatitis in horses, 57
  wound infection, 59
Fowls, *see also* poultry
  anthrax in, 42
  diphtheria in, 260, 261
  epithelioma contagiosum of, 104
  psittacosis in, 153
  rabies in, 137
  "fowl typhoid," 158, 162
Foxes
  contagious abortion in, 75
  rabies in, 134
  trichinosis in, 177
  tularemia in, 213
Frank's, tuberculosis, 2
"French" disease (tuberculosis and syphilis), 2
Friedmann bacillus, 5
Frogs
  botulism in, 277
  diphtheria in, 264
  rabies in, 134, 138
  tuberculosis, 4

G

Gærtner bacillus, 159
Gaertner group in food poisoning, 155, 157, 158
Gas gangrene, 249, 285
  in animals
  in cattle, 287, 294
  in dogs, 287
  in guinea pigs, 287
  in man, 285, 287
  in mice, 287
  in rabbits, 287
  in sheep, 287
  Clostridium fallax, 287
  Clostridium oedematiens, 287
  Clostridium oedematis-maligni, 286
  Clostridium welchi, 285
  prevention of, 288
  symptoms of, 287
Gas oedema bacillus, *see* Clostridium oedematiens
Geese
  actinomycosis in, 94, 96
  plague in, 195, 201
  trichinosis in, 177
Gemara, tuberculosis, 2
Geographical prevalence
  contagious abortion, 67
  foot-and-mouth disease, 55
  malta fever, 67
  plague, 195
  rabies, 121
  rocky mountain spotted fever, 238
  tularemia, 211
Gibraltar fever, see malta fever
Giraffe,
  foot-and-mouth disease, 55
Glanders
  in animals, 112
  in cats, 112, 299
  in dogs, 112, 298
  in donkeys, 112
  in ferrets, 112
  in goats, 112
  in guinea pigs, 112
  in horses, 110, 112, 296
  in man, 113, 296
  in mice, field, 112
  in mice, house, 112
  in mice, white, 112
  in moles, 112
  in mules, 112
  in rabbits, 112
  in sheep, 112
  in swine, 112, 297
  agglutination test for, 116
  complement fixation test for, 116
  control of, 115
  cure of, 114
  diagnosis of, 115
  "farcy buds," 113
  first systematic description of, 110
  from horse to donkey, 110
  history of, 110
  mallein reaction, 115
  mallein test for, 116
  mode of infection, 114
  occupational disease, 113
  prevalence of, 111
  prevention of, 114
  resistance of in man, 114

  Strauss reaction, 115, 116
  symptoms of, 112
  transmissible to man, 110
  vaccination against, 115
Glanders bacillus, 111
  characteristics of, 111
  resistance of, 111
Glanders and farcy, 110
Glandular type of tick fever, *see* tularemia
Goat
  actinomycosis in, 94
  anthrax in, 42, 297
  botulism in, 276
  Brucella melitensis, 297
  contagious abortion in, 75
  cowpox in, 104
  diphtheria in, 263
  foot-and-mouth disease, 55, 297
  glanders in, 112
  malta fever in, 62, 69
  rabies, 297
  rocky mountain spotted fever in, 241
  scarlet fever in, 269
  sheeppox in, 104
  swine-pox in, 104
  tetanus in, 280
  trembles in, 91
  tuberculosis, 6, 7
  tuberculosis, bovine, 297
  tuberculosis, human, 271
  tularemia in, 213
Goat fever, *see also* malta fever
Goat fever, *see also* undulant fever, 78
Goat-pox, 104
Goat-pox
  in man, 104
Goose-pox, 104
Gophers, plague in, 195, 201
Goths, tuberculosis, 1
Ground squirrels
  plague in, 200, 203, 204, 206, 207
  rocky mountain spotted fever, 302
  tularemia in, 212, 214, 217
Grouse
  trichinosis in, 177
  tularemia in, 213, 214
Guarniéri bodies, 101
Guinea pigs
  actinomycosis in, 94
  aertrycke infection in, 161
  anthrax in, 42
  diphtheria in, 261, 263
  foot-and-mouth disease, 56
  gas gangrene in, 287
  glanders in, 112
  malta fever, 70
  plague in, 195
  psittacosis in, 153
  rabies in, 135
  rocky mountain spotted fever in, 241
  S. aertrycke in, 165
  scarlet fever in, 269
  spirochetal jaundice in, 225, 227
  swine erysipelas in, 171
  tetanus in, 279, 281
  trembles in, 91,
  trichinosis in, 177
  tuberculosis, 6, 8
  tuberculosis, avian, 24
  tuberculosis, human, 271
  tularemia in, 213, 215, 217
  vaccinia in, 104
Guinea worm, 298
  in dogs, 298
  in man, 298

H

Hare
  plague in, 302
  tularemia in, 209, 214, 302
Haemodipsus ventricosus, *see* rabbit louse
Hemorrhagic septicemia, in cattle, 294
Hens, *see also* poultry
  malta fever, 70
Hesperomys pulustris, *see* rats
Hindus, tuberculosis, 1
History
  of actinomycosis, 94
  of anthrax, 39
  of contagious abortion, 63
  of cowpox, 100
  of food poisoning, 156
  of foot-and-mouth disease, 53
  of glanders, 110
  of malta fever, 63
  of milk sickness, 86
  of plague, 191
  of psittacosis, 152
  of rabies, 118
  of rat-bite fever, 231

  of rocky mountain spotted fever, 236
  of septic sore throat, 250
  of smallpox, 100
  of swine erysipelas, 170
  of trembles, 86
  of tuberculosis, 1
  of tularemia, 209
  of undulant fever, 63
  of vaccination, 101

Hog, *see* swine

Hog-cholera group in food poisoning, 158

Hoplopsyllus anomalus, *see* fleas, 202

Horses, 294
  actinomycosis in, 94, 96, 296
  anthrax in, 42, 296
  botulism in, 276
  Brucella abortus, 296
  Brucella melitensis in, 296
  contagion, 295
  contagious pneumonia, 295
  cowpox in, 104
  diphtheria in, 261, 263
  dourine, 295
  foot-and-mouth disease in, 56, 296
  glanders in, 110, 112, 296
  horsepox, 295
  infectious abortion, 295
  infectious anemia, 295
  influenza, 295
  malta fever, 70
  osteoporosis, 295
  plague in, 195, 201, 296
  poliomyelitis in, 247
  purpura hemorrhagica, 295
  rabies in, 134, 135, 296
  rocky mountain spotted fever in, 241, 296
  Salmonella abortus-equi, 75, 165
  smallpox in, 104
  sporotrichosis in, 295
  stomatitis, 296
  strangles, 295
  surra, 295
  swine erysipelas in, 171
  tetanus in, 279, 280, 282
  trembles in, 90
  trichinosis in, 177
  tuberculosis, 6, 7, 296
  tuberculosis, avian, 23
  tuberculosis, human, 271
  tularemia in, 213, 296

Horsepox, 104
  in animals
  in cattle, 104
  horses, 295
  in man, 104

Human Diseases spread by Animals, 247

Hydrophobia, *see* rabies

I

Identity
  of smallpox and cowpox, 103

Incubation period
  of rabies in domestic animals, 136
  of rabies in man, 138
  in rat-bite fever, 233
  in septic sore throat, 256

Infectious anemia, horses, 295

Infectious aphtha, *see* foot-and-mouth disease

Infectious abortion in horses, 295

Infectiuos agent
  in plague, 194
  of rabies, 126
  of tularemia, 209, 211, 213

Infectious jaundice, 222
  see also spirochetal jaundice

Infectious ophthalmia, in cattle, 294

Influenza
  horses, 295

Intermediate group in food poisoning, 158

Intestinal flukes
  fasciolopsis of hogs, 185
  paramphistomum cervi in cattle, 185

Intestinal protozoa
  Entamoeba histolytica, 301
  Giardia, 301

Immunization against tuberculosis, 35

J

Johne's bacillus, 5
  disease, 5

Jackals, rabies in, 134

Jimmy weed, *see* rayless goldenrod

K

Kangaroos, plague in, 195

L

Llama
  foot-and-mouth disease, 55
  plague in, 195
Leptospira, 222
Leptospira icteroides, 224
Leptospira icterohemorrhagiæ, 222, 223, 224, 225, 226, 228
  characteristics of, 223
  resistance of, 223, 224
  see also spirochetal jaundice
Lepus campestris, *see* white tailed jack rabbit
Lesions in plague, 199
Lizards
  actinomycosis in, 94, 96
  tuberculosis, 4
Loeffleria nevermanii, *see* foot-and-mouth disease, 57
Lumpy jaw, *see* actinomycosis
Lynx, rocky mountain spotted fever, 302

M

Malignant catarrh, in cattle, 294
Malignant edema, in cattle, 294
Malignant pustule, *see* anthrax
Mallein, discovery of, 111
Malta fever, 62
  agglutination test for, 63
  in animals, 69
  in cats, 70
  in cattle, 70
  in chickens, 70
  in dogs, 70
  in ducks, 70
  in goats, 62, 69
  in guinea pigs, 70
  in hens, 70
  in horses, 70
  in man, 62, 70, 76
    symptoms of, 76
    symptoms of anatomical, 76
    symptoms of bacteriological, 76
    symptoms of clinical, 76
  in mice, 70
  in monkeys, 70
  in mules, 70
  in poultry, 70
  in rabbits, 70
  in rats, 70
  in sheep, 70
  confused with other diseases, 62, 77
  epidemiology of, 63
  from goats milk, 63
  geographical prevalence of, 67
  history of, 63
  seasonal prevalence, 69
  symptoms, 62, 69, 76
Malta fever and contagious abortion, 62
  infecting organisms, table of, 65
Maltese fever, *see* Malta fever
Mammals, tetanus in, 279
Mammitis
  in cattle, 253
  in cows *see* cattle
Mange, in dogs, 298
Man
  abortus bacillus, 76
  actinomycosis in, 94, 96
  aertrycke infection in, 161
  anthrax in, 44
  ascaris lumbricoides in, 181, 185, 186
  botulism in, 275, 276, 277
  Brucella abortus, 76
  Brucella melitensis, 76
  contagious abortion in, 62, 76
  diphtheria in, 260, 261, 262
  dog tapeworm in, 184
  enteriditis in, 160
  fish tapeworm in, 184, 186
  flukes in, 185
  foot-and-mouth disease in, 59
  gas gangrene in, 285, 287
  glanders in, 113, 296
  goat-pox in, 104
  guinea worm, 298
  horsepox in, 104
  malta fever in, 62, 70, 76
  milk sickness in, 91
  plague in, 195, 203
  psittacosis in, 153
  rabies in, 134, 135, 138
    incubation period of, 138
    symptoms of, 138
  rat-bite fever in, 231, 233, 234
  rocky mountain spotted fever in, 236, 241
  scarlet fever in, 266
  septic sore throat in, 250, 254, 255, 256
  smallpox in, 271

  spirochetal jaundice in, 222, 227
  swine erysipelas in, 172, 173
  swine-pox in, 104
  tapeworm in, 181, 182, 186
  tetanus in, 249, 279, 280, 281, 282, 283
  trichinosis in, 177, 178
  tuberculosis in 1, 2, 3, 6, 13, 25, 27, 31
  tularemia in, 209, 212, 215, 217
  undulant fever, 297
  white diarrhea in, 162
  resistance of to glanders, 114
  susceptibility to infection with Brucella melitensis, 83
  symptoms of rabies in, 138
Marmota flaviventor, *see* woodchuck
Mastitis, in cattle, 253
Meat, transmission of tubercle bacilli, 20
"Measly" beef, 183
  see tapeworm
Mediterranean fever, *see* Malta fever
"Mediterranean remittent fever," 63
Melitensis A, 67
Melitensis B, 66
Melitensis-abortus group
  serological classification, 66
  variety abortus, 66
  variety melitensis A, 67
  variety melitensis B, 67
  variety para-abortus, 67
  variety paramelitensis, 67
Melitensis organism
  characteristics of, 66
Melitensis septicemia, *see* malta fever
Micrococcus melitensis, 76
  malta fever, 64, 65
Mice
  aertrycke infection in, 161
  anthrax in, 42
  botulism in, 277
  diphtheria in, 263
  gas gangrene in, 287
  malta fever, 70
  plague in, 195, 201
  psittacosis in, 153
  scarlet fever in, 269
  spirochetal jaundice in, 223
  tetanus in, 281
  trichinosis in, 177
  tuberculosis, 6, 8
  tularemia in, 213, 214, 217
  tularemia, 301
  vaccinia in, 104
Mice, field, glanders in, 112
Mice, house, glanders in, 112
Mice, white
  glanders in, 112
  rocky mountain spotted fever in, 241
Milk, transmission of tubercle bacilli, 16
Milk sickness, 91
  see also trembles
  in animals
  in man, 86
    source of infection, 91
    symptoms of, 91
  swine, 297
  from poisonous plants, 87
  history of, 86
  prevention of, 92
  source of infection, 86
Milk sick,*see* milk sickness
Mishna, tuberculosis, 2
Mist bacillus, 5
Moles, glanders in, 112
Monkey
  actinomycosis in, 94, 96
  cowpox in, 104
  diphtheria in, 263
  malta fever, 70
  plague in, 195
  rabies in, 135
  rocky mountain spotted fever in, 241
  scarlet fever in, 269
  smallpox in, 103, 104
  S. suipestifer in, 165
  trembles in, 91
  tuberculosis, 6
  tuberculosis, human, 271
  tularemia in, 212
  vaccinia in, 104
Mortality
  from tetanus, 283
  in plague, 194
  in psittacosis, 153
  in rabies, 138
  in spirochetal jaundice, 225
  in trichinosis, 178

Mountain fever, *see* malta fever
Mountain fever, *see* Rocky Mountain Spotted Fever
Mouse, actinomycosis in, 94
Mouse, white, avian tuberculosis, 23
Mouse louse, 216
Mules
  glanders in, 112
  malta fever, 70
  rabies in, 134
  tuberculosis, 8
Murrain, *see* anthrax
Mus musculus, *see* rats, 201
Mus norvegicus, spirochetal jaundice, 223
Musk shrew, plague in, 302
Mycobacterium
  alluvialum, 5
  avium, 3
  berolinensis, 5
  butyricum, 5
  chelonei, 4
  friburgensis, 5
  graminis, 5
  paratuberculosis, 5
  phlei, 5
  piscium, 4
  ranæ, 4
  smegmatis, 5
  stercusis, 5
  tropidonatum, 4
  tuberculosis (bovis), 3
  tuberclulosis (hominus), 3

N

Nagana, in cattle, 294
Neapolitan fever, *see* malta fever
Necrotic stomatitis, in cattle, 294
Negri bodies, 120, 126, 127, 128
Neosocia bandicota, *see* rats, 201
Neotoma, *see* brush rat, 201
"Neurocytes hydrophobiæ," *see* rabies, 127
Norway rat plague, 198

O

Ohara's disease, *see* tularemia
Olives, botulism in, 277
Opisthorchis felineus, *see* flukes
Opisthorchis pseudofelineus, *see* flukes
Opossum, tularemia in, 213, 214
Osteoporosis, in horses, 295

P

Paraabortus, 67
Paramelitensis, *see* melitensis, 66
Paratyphosus B (porcine), in swine, 164
Parrakeets, psittacosis in, 152, 153
Parrots
  psittacosis in, 152, 153, 302
  tuberculosis, 6, 8, 302
  tuberculosis, human, 271
Parsees, tuberculosis, 1
Paschen bodies, 102
Pasteurella muriseptica, *see* septicemia in mice
Pasteurella pestis, 191
  in plague, 191
Pasteurella tularensis, *see* Bacterium tularense
Pasteurization of milk, 30, 31
Pearly disease (tuberculosis), 1, 11
Pesta magna, *see* small pox
Pestis caviae, in rats, 161
Pfeifferella mallei, 111
Phthisis (tuberculosis), 1
Pig, *see* swine
Pigeons
  rabies in, 137
  scarlet fever in, 269
  swine erysipelas in, 171, 172
  trichinosis in, 177
  tuberculosis, avian, 24
  tularemia in, 213
Pigeon-pox, 104
Pine squirrel, rocky mountain spotted fever in, 240
Plague, 191
  in the ancients, 191
  animals susceptible to, 195
  in animals, 198
  in bats, 195, 201
  in booby owl, 201
  in California ground squirrel, 302
  in camels, 195, 201
  in cats, 195, 201, 299
  in cattle, 195, 201
  in chickens, 195, 201
  in dogs, 195, 201
  in donkeys, 195, 201
  in fleas, 202
    cat flea, 202

    dog flea, 202
    European rat flea, 202
    human flea, 202
    Indian rat flea, 202
    mouse flea, 202
    squirrel flea, 202
    tarbagan flea, 202
  in ferrets, 201
  in geese, 195, 201
  in gophers, 195, 201
  in ground squirrels, 200, 203, 204, 206, 207
  in guinea pigs, 195
  in hare, 302
  in horses, 195, 201, 296
  in kangaroos, 195
  in llamas, 195
  in man, 195, 203
    symptoms of, 203
    types of, 203
  in mice, 195, 201
  in monkeys, 195
  in musk shrew, 302
  in pigeons, 201
  in prairie dogs, 195, 201
  in rabbits, 195, 302
  in rats, 195, 201, 301
    Alexandrian rat, 198
    bandicot rat, 201
    brush rat, 201
    English rat, 198
    field rat, 201
    Norway rat, 198
    destruction of, 205, 207
  in reptiles, 195
  in rodents, 191, 198, 200, 201, 204
  in sheep, 195, 201
  in squirrels, 195
  in swine, 195, 201
  in tarbagans, 195, 200, 302
  in weasels, 195, 201
  in wood chucks, 195, 201
  in veldt rodents
    dwarf mouse, 201
    fat mouse, 201
    gerbillie or springhaasrot, 201
    ground squirrel, 201
    hares, 201
    karroo rats, 201
    long-eared mouse, 201
    molerat, 201
    multi-mammate mouse, 201
    namaqua gerbillie, 201
    springhare, 201
    striped mouse, 201
    suricate, 201
    water rat, 201
    white-tailed rat, 201
    yellow mongoose, 201
  bacillus, 282
  bacillus pestis, 194
  control of, 201
  geographical prevalence of, 195
  history of, 191
  in hot climates, 195
  in India, 194
  infectious agent, 194
  lesions in, 199
  mortality of, 194
  optimum temperature, 198
  pasteurella pestis, 191
  prevention of, 205-207
  seasonal prevalence of, 197, 204
  source of infection, 203
  vaccine against, 206
  without lesions, 199
Plague-like disease of rodents, *see* tularemia
Pocket gopher, tularemia in, 213
Polewort, *see* white snake root
Poisonous plants
  milk sickness
    rayless goldenrod, 87, 88
    white snake root, 87, 88
Poliomyelitis
  in animals
  in cats, 247
  in cattle, 248
  in chickens, 247, 248
  in dogs, 247, 248
  in horses, 247
  in rabbits, 248
  in swine, 247
Polyplax serratus, *see* mouse louse
Porcine, *see* Swine, contagious abortion, organism-characteristics of, 66
Porcupine, tularemia in, 213
Pork tapeworm (Taenia solium), 183, 186
Poultry
  botulism in, 276
  diphtheria in, 302
  foot-and-mouth disease, 56
  malta fever, 70
  swine erysipelas in, 172
  trembles in, 91
  tuberculosis, 6, 8
  tuberculosis, avian, 20, 301
  white diarrhea, 301

  vaccinia in, 104
  elimination of tuberculosis in, 29
Prairie dogs
  plague in, 195, 201
  rabies in, 135
Prevalence
  of food poisoning, 156
  of glanders, 111
  of septic sore throat, 254
Prevention
  of actinomycosis, 97
  of ascaris infection, 181
  of botulism, 277
  of food poisoning, 166
  of gas gangrene, 288
  of glanders, 114
  of milk sickness, 92
  of plague, 205-207
  of psittacosis, 153
  of rabies, 139
  of rat-bite fever, 233
  of rocky mountain spotted fever, 242
  of scarlet fever, 269
  of septic sore throat, 257
  of spirochetal jaundice, 227
  of tapeworm, 183
  of tetanus, 283
  of trembles, 91
  of trichinosis, 179, 180
  of tularemia, 218
Production of vaccine virus, 105
Protective power of vaccine virus, 106
Protozoan theory of rabies, 127
Psittacosis, 152
  in animals, 152, 153
  in fowl, 153
  in guinea pigs, 153
  in man, 153
  in mice, 153
  in parrakeets, 152, 153
  in parrots, 152, 153, 302
  in rabbits, 153
  causative agent
    characteristics of, 152
    Salmonella psittacosis, 152, 153
  history of, 152
  mortality of, 153
  prevention of, 153
  source of infection, 153
  symptoms of, 153
"Ptomaine poisoning," *see* food poisoning, 155
Puking fever, *see* milk sickness
Pulex irritans, *see* fleas, 202
Purpura hemorrhagica, horses, 295

R

Rabbits
  aetinomycosis in, 94
  aertrycke infection in, 161
  anthrax in, 42
  botulism in, 277
  cowpox in, 104
  diphtheria in, 263
  foot-and-mouth disease, 56
  gas gangrene in, 287
  glanders in, 112
  malta fever, 70
  plague in, 195, 302
  poliomyelitis in, 248
  psittacosis in, 153
  rabies in, 134, 135
  rocky mountain spotted fever in, 241
  scarlet fever in, 269
  smallpox in, 104
  spirochetal jaundice in, 225
  swine erysipelas in, 171
  tetanus in, 279, 281
  trichinosis in, 177
  tuberculosis, 6, 8
  tuberculosis, avian, 23
  tuberculosis, human, 271
  tularemia in, 209, 212, 214, 215, 302
  vaccinia in, 104
  vaccinia in the brain of, 104
  vaccine virus in, 102
  white diarrhea in, 162
Rabbit fever, *see* tularemia
Rabbit louse, 215
Rabies, 118
  amongst the ancients, 118
  in animals, 134
  in asses, 134
  in birds, 137
  in cats, 134, 135, 137, 299
  in cattle, 134, 135, 137, 294
  in chickens, 135
  in children, 139
  in dogs, 134, 135, 136, 137, 298
    elimination of, 143
    in Illinois, 148
    vaccination against, 145
  in fowl, 137
  in foxes, 134
  in frogs, 134, 138
  in goats, 297
  in guinea pigs, 135

in horses, 296
in jackals, 134
in man, 134, 135, 138
in man sam as in dog, 119
in monkeys, 134, 135
in mules, 134
in pigeons, 137
in prairie dogs, 135
in rabbits, 134, 135
in rats, 135, 137
in sheep, 134, 135, 297
in skunks, 134, 135, 137, 298
in squirrels, 135
in stray dogs in Illinois, 148
in swine, 134, 135
in tortoise, 138
in turkeys, 135
in turtles, 134
in wolves, 134, 135
anti-rabic vaccination, 141
anti-rabic vaccination in Illinois, 1928, 147
care of suspected dogs, 141
cauterizing wounds, 143
decrease in, 146
diagnosis of, 120
during the middle ages, 119
"fixed virus," 126, 127
geographical prevalence of, 121
history of, 118
incubation period in domestic animals, 136
infectious agent, 126
mortality in, 138
nature of the Negri bodies, 128
negri bodies, 120, 126, 127,128
prevention of, 139
protozoan theory, 127
seasonal prevalence of, 121
statistics on world prevalence of, 124, 126
"street virus," 126
"street virus" distinct from other viruses, 129
symptoms of, 119
symptoms in dogs, 136
treatment of, 139
treatment of dog bites, 142
vaccinated dogs in U. S., 147
vaccination against, 120, 129
  methods of Cumming, 133
  methods of Harris, 132
  methods of Hygienic Laboratory, 130
  methods of Högyes, 130
  method of Japanese, 145
  methods of Pasteur, 129
  method of Semple, 133
virulence of saliva from a rabid dog, 119
Rabies vaccine, 129
Rabies virus
  action on of gastric juice, 149
  in milk, 149
  in meat, 149
  resistance of, 126
  transmitted through milk, 149
  variation of virulence in, 129
Rats
  black rat, 300
  brown rat, 300
  roof rat, 300
  aertrycke infection in, 161
  botulism in, 277
  diphtheria in, 263
  enteriditis in, 160
  foot-and-mouth disease, 56
  malta fever, 70
  pestis-caviae in, 161
  plague in, 195, 201, 301
  rabies in, 135, 137
  rat-bite fever in, 231-234, 301
  salmonella enteriditis in, 301
  scarlet fever in, 269
  spirochetal jaundice in, 222-225, 227, 228
  tetanus in, 281
  trichinosis in, 175, 176, 177, 301
  tuberculosis, 6, 8
  tuberculosis, avian, 24
  tularemia in, 213, 214, 217, 301
  vaccinia in, 104
  malta fever, 70
Rats, white, rocky mountain spotted fever in, 241
Rats, wild
  S. aertrycke in, 165
  S. enteriditis in, 165
Rat-bite fever, 231
  in animals, 232
  in cats, 231, 232, 299
  in dogs, 233, 299
  in ferret, 231, 233
  in man, 231, 233, 234
  in rats, 231, 234, 301
  in squirrel, 231, 233
  in weasels, 231, 233
  causative agent, 232
  history of, 231

  incubation period, 233
  prevention of, 233
  Spirocheta morsus muris, 232
  symptoms of, 233
Rattus norvegicus, plague, 198
Rattus rattus alexandrinus, plague, 198
Rattus rattus rattus, plague, 198
Rayless goldenrod (Aplopappus heterophyllus), milk sickness, 87-88
"Remittent malarial fever," *see* undulant fever, 63
Reptiles, plague in, 195
Resistance of
  glanders bacillus, 111
  rabies virus, 126
  tetanus organism, 279
  trichinae, 180
Rinderpest in cattle, 294
Ringworm in dogs, 298
Rich weed, *see* white snake root
Rio Grande fever, *see* malta fever
Rock fever, *see* malta fever
Rodents
  plague in, 191, 198, 200, 201, 204
  rocky mountain spotted fever in, 236, 240
  tularemia in, 209, 214
Rocky mountain goat, rocky mountain spotted fever, 302
Rocky Mountain Spotted Fever, 236
  in animals, 240
  in badger, 241, 302
  in bear, 302
  in cattle, 241
  in Columbian ground squirrel, 240
  in cottontail rabbit, 241
  in coyote, 302
  in goats, 241
  in ground squirrel, 302
  in guinea pigs, 241
  in horses, 241, 296
  in lynx, 302
  in man, 236, 241
  in mice, white, 241
  in monkeys, rhesus, 241
  in pine squirrel, 240
  in rabbits, domestic, 241
  in rats, white, 241
  in Rocky mountain goat, 302
  in rodent, 236, 240
  in sheep, 241
  in side-stripped ground squirrel, 240
  in white bellied chipmunk, 240
  in white tailed jack rabbit, 241
  in wood-chuck, 240, 302
  in yellow bellied chipmunk, 240
  causative agent, 238
  history of, 236
  geographical distribution, 238
  prevention of, 242
  seasonal prevalence of, 238
  symptoms of, 241
  ticks
    Amblyomma americanum, 240
    Dermacentor albipictus, 240
    Dermacentor andersoni, 240
    Dermacentor modestus, 240
    Dermacentor occidentalis, 240
    Dermacentor variabilis, 240
    Haemaphysalis, leporis-palustris, 240
  Dermacentroxenus rickettsi, 239
  vaccination against, 242
"Rotlauf," *see* swine erysipelas
"Rouget," *see* swine erysipelas
Round worms, *see also* Ascaris lumbricoides, 181
  in cats, 299
  in swine, 296
  Trichinelle spiralis, 175, 299

S

Salmonella anatum, 158
  in ducks, 165
Salmonella abortus-equi, 158
Salmonella abortus-equi, abortion in horses, 295
  contagious abortion in mares, 162
  in horses, 75, 165
Salmonella aertrycke, *see also* (Bacillus aertrycke), 161, 297
  baboons, 302
  in guinea pigs, 165
  in sheep, 164, 297
  in skunk, 165
  in rats, wild, 165
Salmonella enteriditis, 158, 159, 205
  in animals,
  in cats, 165
  in dogs, 165
  in swine, 164
  in rats, 301
  in rats, wild, 165

Salmonella gallinarum, (Bacillus sanguinarum)
  in chickens, 165
  in "fowl typhoid," 158, 162
Salmonella group
  in food poisoning, 155, 157, 158
  table of, 166
Salmonella pestis-caviae, 161, 165
Salmonella paratyphi, 158
Salmonella psittacosis, 158
Salmonella pullorum, 158, 301
Salmonella pullorum
  in chickens, 165
  white diarrhea in chickens, 158, 162
Salmonella schottmülleri, 158, 161
Salmonella suipestifer, 158, 159, 297
  in animals,
  in monkey, 165
  in swine, 164, 297
Salmonella typhimurium, 205
Sarcoma of the jaw, *see* actinomycosis
Scarlet fever, 266
  in animals,
  in cats, 269
  in cattle, 266, 269, 294
  in chickens, 269
  in chimpanzees, 269
  in cows, *see* cattle
  in dogs, 269, 299
  in goats, 269
  in guinea pigs, 269
  in man, 266
  in mice, 269
  in monkeys, 269
  in pigeons, 269
  in rabbits, 269
  in rats, 269
  in sheep, 269
  in swine, 269
  prevention of, 269
Sciurus hudsonicus, *see* pine squirrel
Scrofula (tuberculosis), 1
Seasonal prevalence
  of contagious abortion, 69
  of malta fever, 69
  of plague, 197, 204
  of rabies, 121
  of rocky mountain spotted fever, 238
  of tularemia, 211
Septic sore throat, 250
  in animals, 253
  in cattle, 250, 253, 254, 255, 294
  in man, 250, 254, 255, 256
  history of, 250
  incubation period, 256
  prevalence of, 254
  prevention of, 257
  symptoms of, 256
Sheep
  aertrycke infection in, 161
  actinomycosis in, 94, 96
  anthrax in, 42
  contagious abortion in, 75
  cowpox in, 104
  diphtheria in, 263
  dog tapeworm in, 185, 297
  flukes in, 185, 297
  food poisoning in, 164
  foot-and-mouth disease, 55, 297
  gas gangrene in, 287
  glanders in, 112
  malta fever, 70
  plague in, 195, 201
  rabies in, 134, 135, 297
  rocky mountain spotted fever in, 241
  Salmonella aertrycke in, 164, 297
  scarlet fever in, 269
  smallpox in, 104
  swine erysipelas in, 171
  swine-pox in, 104
  tetanus in, 280
  trembles in, 90
  trichinosis in, 177
  tuberculosis, 6, 7, 297
  tularemia in, 213, 214
Sheep-pox (variola ovina), 104
  in goats, 104
Side-stripped ground squirrel, rocky mountain spotted fever in, 240
Skunks
  S. aertrycke in, 165
  rabies in, 134, 135, 137, 298
Slows, *see* milk sickness
Slow fever, *see* malta fever
  see undulant fever, 28
Smallpox and cowpox, 100
  identity of, 103
Smallpox
  in animals
  in cattle, 271
  in horses, 104
  in man, 100, 271
  in monkeys, 103, 104
  in sheep, 104

  in rabbits, 104
  deaths from, 106
  etiological agent, 101
  history of, 100
Smallpox and cowpox
  relationship between, 102
Smallpox
  transmission of, 103
  in U, S., 1921-26, 107
Smegma bacillus, 5
Snail
  flukes in, 185
Snakes
  tuberculosis, 4
"Sodoku" *see* rat-bite fever
Source of infection
  of botulism, 275, 276
  of enteriditis, 160
  of plague, 203
  of psittacosis, 153
  of tetanus, 280
  of trichinosis, 176, 177
Sparrow
  avian tuberculosis, 24
Spinach
  botulism in, 277
Spirocheta icterohemorrhagiae, 222, 302
Spirocheta morsus muris
  rat-bite fever, 232
Spirocheta nodosa, 222
Spirochetal jaundice, 222
  in dogs, 225
  in guinea pigs, 225, 227
  in man, 222-227
  in mice, 223
  in rabbits, 225
  in rats, 222-225, 227-228
  Mus alexandrinus,
    decumanus, 224
    norvegicus, 224
  infectious agent, 223
  mortality in, 225
  prevention of, 227
  source of infection, 227
  symptoms of, 225
  test for, 224
  in U. S., 226
  vaccination against, 228
Spirochetosis icterohemorrhagiae, *see* spirochetal jaundice
Splenic fever, *see* anthrax
Sporotrichosis
  in horses, 295
Sporozoa muris, 232
Spotted Fever, *see* Rocky Mountain Spotted Fever
Squaw weed, *see* white snake root
Squirrels
  plague in, 195
  rabies in, 135
  rat-bite fever in, 231, 233
Squirrel flea, 216
Stable fly, 216
Staphylococcus albus, 253
Staphylococcus aureus, 253
Statistics on world prevalence
  of rabies, 124-126
Stomatitis
  horses, 296
Stomoxys calcitrans, *see* stable fly
Strains
  of abortus, 66
  of melitensis, 66
Strangles
  horses, 295
"Street virus," *see* rabies
Streptococcus
  agalactiae contagiosae, 251
  asilignus, 252
  epidemicus, 250-254, 257, 258
  epidemicus
    method of detection in milk, 252
  hemolyticus α (alpha), 251
  hemolyticus β (beta), 251
  hemolyticus i, 252
  infrequens, 252
  mastiditis, 251, 252
  mastitis, 251
Streptotrix muris ratti, 232
Strepticoccus pyogenes, 251
  pyogenes bovis, 251
  scarlatinae, 266, 269
  subacidus, 252
String beans
  botulism in, 277
Suipestifer, infection, 159
Superfamily Trichuroidea, 175
Surra
  in horses, 295
Susceptibility of animals to Br. melitensis and Br. abortus, table of, 69
Swine
  actinomycosis in, 94, 96, 297
  aertrycke infection in, 161
  anthrax in, 42, 297
  ascaris suis in, 181, 186
  B. paratyphosus B. (porcine) in, 164

B. typhi suis in, 164
botulism in, 276
contagious abortion in, 62, 75, 297
food poisoning in, 164
foot-and-mouth disease in, 55, 297
glanders in, 112, 297
milk-sickness, 297
plague in, 195, 201
poliomyelitis in, 247
rabies in, 134, 135
round worm, 296
S. enteriditis in, 164
S. suipestifer in, 164, 297
scarlet fever in, 269
suipestifer-voldagsen in, 164
swine erysipelas, 297
swinepox in, 104
tapeworm in, 181, 182, 183, 186, 296
trembles in, 91
trichinosis in, 175, 176
tuberculosis, 6, 7, 25
tuberculosis, avian, 23, 25, 26, 271
tuberculosis, bovine, 25, 26, 271, 296
tuberculosis. human, 26, 271, 296
tuberculosis from milk, 25
tularemia in, 213, 217
Swine erysipelas (Erysipelas rhusiopathiae), 170
Swine erysipelas
in animals, 171
in cattle, 171
in guinea pigs, 171
in horses, 171
in man, 172, 173
in pigeon, 171, 172
in poultry, 172
in rabbit, 171
in sheep, 171
swine, 297
causative agent, 170, 171
history of, 170
symptoms of, 172
Swine-pox (variola suilla), 104
in animals
in goats, 104
in man, 104
in sheep, 104
in swine, 104
Syphilis
confused with tuberculosis, 2
cure of by rat-bite fever, 233
Symptoms
of contagious abortion, 73
of gas gangrene, 287
of malta fever, 62, 69, 76
of plague, 203
of psittacosis, 153
of rabies, 119
of rat-bite fever, 233
of rabies in dogs, 136
of rabies in man, 138
of rocky mountain spotted fever, 241
of septic sore throat, 256
of spirochetal jaundice, 225
of trichinosis, 178
of swine erysipelas, 172
Sylvilagus nuttali, *see* Cottontail rabbit

T

Taenia echinococcus, 184, 186, 297, 298
dog tapeworm, 184, 186
Taenia saginata, 294
beef tapeworm, 181, 182, 186
Taenia solium, 296
pork tapeworm, 181, 182, 183, 186
Talmud
tuberculosis, 2
Tapeworms, 182-186
in animals
in dogs, 184, 186, 298
in fish, 182, 183, 184, 186
in man, 181, 182, 186
in swine, 181, 182, 183, 186, 296
beef tapeworm, 182, 186
Dipylidium caninum, 185, 299
pork tapeworm, 181, 182, 183, 186
prevention of, 183
Tarbagans
plague in, 195, 200, 302
Taxidea taxus, *see* badger
Tetanus
in animals, 249, 279
in cats, 281
in cattle, 279, 280, 294
in chicken, 281
in dogs, 281
in goats, 280
in guinea pigs, 279, 281
in horses, 279, 280, 282
in mammals, 279
in man, 249, 279, 280, 281, 282, 283
in mice, 281
in new born, 282
in rabbits, 279, 281
in rats, 281
in sheep, 280

  antitoxin, 281
  mortality, 283
  prevention of, 283
  resistance of, 279
  source of infection, 280
  vaccination against, 282
Texas fever
  in cattle, 294
Ticks, 215, 240, 302
  Dermacentor, 302
  woodtick, 215, 302
Toads
  tuberculosis, 4
Tortoise
  rabies in, 138
Transmission
  of smallpox, 103
Treatment
  of dog bites, 142
  of rabies, 139
Trematol
  causing milk sickness, 87
  poisonous principle in white snake root, 89
  test for, 89
Trembles, *see also* milk sickness, 86
  in animals, 86
  in cats, 91
  in cattle, 90
  in dogs, 91
  in goats, 91
  in guinea pigs, 91
  in horses, 90
  in monkeys, 91
  in poultry, 91
  in sheep, 90
  in swine, 91
  history of, 86
  prevention of, 91
  symptoms of, 90
Trichinae
  resistance of, 180
Trichina spiralis, 175
Trichinella spiralis, 175, 185, 186, 296
Trichinosis, 175
  in the ancients, 175
  in animals, 175
  in badger, 177
  in bear, 177
  in cats, 177
  in cattle, 177
  in chicken, 177
  in dogs, 177, 299
  in fox, 177
  in geese, 177
  in grouse, 177
  in guinea pigs, 177
  in horses, 177
  in man, 177, 178
  in mice, 177
  in pigeons, 177
  in rabbits, 177
  in rats, 175, 176, 177, 300
  in sheep, 177
  in swine, 175, 176
  confused with, 179
  diagnosis of, 179
  mortality in, 178
  prevention of, 179, 180
  resistance of, to heat, 180
  source of infection, 176, 177
  symptoms of, 178
  test for, 179
Trichuris trichuria, 175
Tubercle bacilli, bovine, human infections, 11
Tuberculin
  cattle, 10
  test, avian, 20
  testing, 18
Tuberculosis
  in animals
  cats, 299
  in cattle, 294
  cold-blooded animals, 4
  in dogs, 298
  horses, 296
  in man, 1, 2, 3, 6, 13, 25, 27, 31
  in parrots, 302
  sheep, 297
  in swine, 25
  in swine, table of, 26
Tuberculosis, avian type, 3, 6, 20, 248
  in man, 24
  in poultry, 301
  in swine, 271
Tuberculosis, bovine, 8, 248
  goats, 297
  in swine, 271
Tuberculosis
  eradication of, in animals, 28
  history, 1
Tuberculosis, human type, 248, 271
  in apes, 271
  in cats, 271
  in cattle, 271
  in cockatoos, 271
  in dogs, 271
  in goats, 271
  in guinea pigs, 271

  - in horses, 271
  - in monkeys, 271
  - in parrots, 271
  - in rabbits, 271
  - in swine, 271
  - immunization against, 35
  - meat inspection, 32
  - meat inspection, causes of contamination, table of, 34
  - prevention and control, 27
  - pulmonary, 248
  - susceptibility to infection, 5
  - tubercle bacilli in meat, 20
  - tubercle bacili in milk, 16
  - thermal death point of tubercle bacilli, 31
  - thermal death points of tubercle bacilli, table of, 32
  - types of organisms, 3
  - Yersin type, 24
- Tularemia, 209
  - in animals
  - in cats, 213, 299
  - in cattle, 213
  - in chickens, 213
  - in chipmunk, 213
  - in coyote, 213, 217
  - in dogs, 213
  - in fox, 213
  - in goats, 213
  - in ground-squirrel, 212, 214, 217
  - in grouse, 213, 214
  - in guinea pigs, 213, 215, 217
  - in hares, 209, 214, 302
  - in horses, 213, 296
  - in man, 209, 212, 215, 217
  - in mice, 213, 214, 217, 301
  - in monkey, 212
  - in opossum, 213, 214
  - in pigeons, 213
  - in pocket gopher, 213
  - in porcupine, 213
  - in rabbits, 209, 212, 214, 215, 302
  - in rats, 213, 214, 217, 301
  - in rodents 209, 214
  - in sheep, 213, 214
  - in swine, 213, 217
  - in turkey, 213
  - in wood chuck, 213
  - confused with, 218
  - geographical prevalence, 211
  - history of, 209
  - infectious agents, 209, 211, 213
  - prevention of, 218
  - seasonal prevalence of, 211
  - sources of infection, 218
  - transmitting agent
    - bed bug, 215
    - deer fly, 215
    - mouse louse, 216
    - rabbit louse, 215
    - squirrel flea, 216
    - stable fly, 216
    - tick, 215
    - woodtick, 215
  - treatment of, 218
  - types of
    - glandular, 218
    - oculoglandular, 218
    - typhoid, 218
    - ulceroglandular, 218
- Turkeys
  - rabies in, 135
  - tularemia in, 213
- Turtles
  - rabies, 134
  - tuberculosis, 4
- Typhoid fever
  - in animals
  - in cattle, 248
  - in chickens, 248
  - in fowl, 158, 162
- Typho-malaria fever of the Rocky Mountains, *see* Rocky Mountain Spotted Fever

U

- Undulant fever, *see* malta fever
  - in animals
  - in adults, 82
  - cats, 299
  - in children, 82
  - man, 76, 297
  - in man of goat origin, world distribution of, 68
  - agglutination tests, 80
  - caprine origin in U. S., 78
  - complement fixation tests, 79
  - epidemic of, 78
  - history of, 63
  - human infection, bovine origin, 80
  - inter-human transmission, 82
  - in laboratory workers, 82
  - pasteurization of milk, 83
  - porcine origin, 82
  - prevention, 82
  - recognized in 1905, 77
  - susceptibility of man to Brucella abortus, 82

V

Vaccinated dogs in U. S., 147
Vaccination, 101
  discovered by, 101
  of dogs against rabies, 145, 146
  of dogs, Japanese method, 145
  history of, 101
Vaccination against
  rabies, 120, 129, 130
  rocky mountain spotted fever, 242
  spirochetal jaundice, 228
  plague, 206
  tetanus, 282
Vaccine virus, 102, 105
  action of disinfectants on, 102
  in rabbits, 102
  production of, 105
  protective power of, 106
  resistance of, 102
Vaccinia, *see* cowpox
Vaccinia, 100, 101
  in animals
  in brain of rabbits, 104
  in cats, 104
  in guinea pigs, 104
  in mice, 104
  in monkeys, 104
  in poultry, 104
  in rabbits, 104
  in rats, 104
  from smallpox, 103
Variation of virulence
  in rabies viruses, 129
Variola, *see* smallpox
Variola anserum, *see* goose-pox
Variola canina, *see* dog pox
Variola caprina, *see* goat-pox
Variola columbarum, *see* pigeon pox
Variola gallinarum, *see* chicken pox
Variola ovina, *see* sheep pox
Variola suilla, *see* swine pox
Variola vaccinia, *see* cowpox
Variola vera, 103
Variola virus, 102
Veda
  tuberculosis, 1
Vibrion septique Pasteur, *see* Clostridium oedematis-maligni

W

Weasels
  plague in, 195, 201
  rat-bite fever in, 231, 233
Weil's disease, *see* spirochetal jaundice
  in chimpanzees, 302
Welch bacillus, 281, 285
White bellied chipmunk
  rocky mountain spotted fever in, 240
White tailed jack rabbit
  rocky mountain spotted fever in, 241
White diarrhea
  in animals
  in cats, 162
  in chickens, 158, 162
  in man, 162
  in poultry, 301
  in rabbits, 162
White snake root
  Eupatorium serotinum and
  Eupatorium urticaefolium
  milk sickness, 87, 88
    experimental feeding of, 91
White snake root poisoning, *see* milk sickness
White scours
  in cattle, 294
Woodchuck
  plague in, 195, 201
  rocky mountain spotted fever in, 240, 302
  tularemia in, 213
Wooden tongue, *see* actinomycosis
Woodtick, 215
Worms, 175-186
Wolves
  rabies in, 134, 135

X

Xenopsylla astia, *see* fleas, 203
Xenopsylla cheapis, *see* fleas, 202, 203

Y

Yellow bellied chipmunk
  rocky mountain spotted fever in, 240

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